Historic, Archive Document

Do not assume content reflects current
scientific knowledge, policies, or practices.

Reserve
aSF771
.C3
1981
5th ED

The Plum Island Animal Disease Center
U.S. DEPARTMENT OF AGRICULTURE
Greenport, New York 11944

ILUSTRATED MANUAL FOR THE RECOGNITION AND DIAGNOSIS OF CERTAIN ANIMAL DISEASES

AD-33 Bookplate
(1-63)

NATIONAL

AGRICULTURAL

LIBRARY

ILLUSTRATED MANUAL FOR THE RECOGNITION AND DIAGNOSIS OF CERTAIN ANIMAL DISEASES

U.S. DEPT. OF AGRICULTURE
NATIONAL AGRICULTURAL LIBRARY

MAR 18 1982

CATALOGING-PREP.

AFRICAN SWINE FEVER
HOG CHOLERA
FOOT-AND-MOUTH DISEASE
VESICULAR STOMATITIS
SWINE VESICULAR DISEASE
VESICULAR EXANTHEMA OF SWINE
BOVINE PAPULAR STOMATITIS
RINDERPEST
MALIGNANT CATARRHAL FEVER
INFECTIOUS BOVINE RHINOTRACHEITIS
BOVINE VIRAL DIARRHEA
CONTAGIOUS BOVINE PLEUROPNEUMONIA
LUMPY SKIN DISEASE
DERMOPATHIC BOVINE HERPES VIRUS INFECTION
AFRICAN HORSE SICKNESS
RIFT VALLEY FEVER
CONTAGIOUS ECTHYMA
SHEEP POX
BLUETONGUE
FOWL PLAGUE
VELOGENIC VISCEROTROPIC NEWCASTLE DISEASE
CONTAGIOUS EQUINE METRITIS

The Plum Island Animal Disease Center
U.S. DEPARTMENT OF AGRICULTURE
Greenport, New York 11944

Fifth Edition
1981

By Drs. Jerry J. Callis,* Ahmed H. Dardiri,* Deam H. Ferris,*
Juan Gay G.,**, John Mason** and Francis W. Wilder,**

*Plum Island Animal Disease Center
**México-U.S. Commission for the Prevention of Foot-and-Mouth Disease

CONTENTS

768553

PREFACE

This Manual is designed to provide a brief summary of a number of animal diseases exotic to most of the Americas, and a number of others that should be considered in the differential diagnosis of some of these diseases, together with pictures of characteristic lesions for each of the different diseases. The primary purpose of the Manual is to serve as a field guide for the recognition and diagnosis of these diseases.

For a more complete description of most of the diseases discussed in this Manual, the Handbook on Foreign Animal Diseases, published by the U.S. Animal Health Association, should be consulted, together with other standard texts.

The Manual is an elaboration of a "Pocket Handbook" first published by the Plum Island Animal Disease Center in 1977, with color illustrations in microfiche.

The Spanish translation of the Manual is the product of a cooperative effort by OIRSA (Organismo Internacional Regional de Sanidad Agropecuaria), CPA (Comisión México-Americana para la Prevención de la Fiebre Aftosa) and DIGSA (Dirección General de Sanidad Animal, México).

The photographs used in this Manual have been obtained from various sources. These include the Plum Island Animal Disease Center, Greenport, NY; the National Veterinary Services Laboratories, Ames, Iowa; the Arthropod-borne Animal Disease Research Laboratory, Denver, Colo.; the APHIS Emergency Programs, Hyattsville, MD; Dr. Frank Ramsey, College of Veterinary Medicine, Iowa State University, Ames, Iowa; and the Grosse Ile Experiment Station, Quebec, Canada.

AFRICAN SWINE FEVER

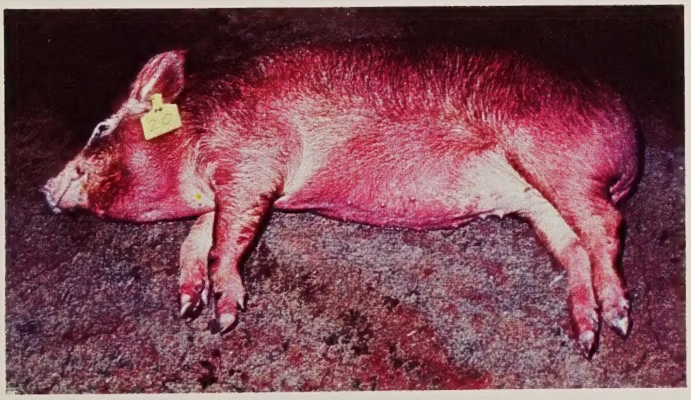
Definition: Classic African swine fever (ASF) is a highly contagious, often acute, viral disease of domestic swine characterized by fever, marked cyanosis of skin areas, and pronounced hemorrhages of the internal organs, particularly the lymph nodes, kidney, and gastrointestinal mucosa. Mortality frequently approaches 100 percent in initial epizootics. (In the classic disease)*

Etiology: The causative agent of ASF is a DNA virus which is 175 to 215 nm in diameter belonging to the family Iridoviridae. It is sensitive to lipid solvents and ortho-phenylphenol disinfectants but is resistant to strong acids and alkalies. The virus causes hemadsorption of swine red cells in infected leukocyte cultures. Inclusion bodies are found within the cytoplasm of cells infected with the virus.

It remains viable at refrigeration temperatures for 18 months. In 1957 the disease appeared in Portugal, presumably imported by accident from Africa. From there the disease spread to Spain. By 1967 it was reported in Italy; and in 1971 it was found in Cuba. In 1978 new foci of ASF appeared in the Dominican Republic, Haiti, Brazil and Sardinia. In 1979 it reappeared in Cuba.

Transmission: Infection is most common as a result of contact with recovered or carrier pigs and ingestion of contaminated or infected garbage, urine, feces, and carcasses. Recently, experimental transmission was achieved in Africa and Spain with infected ticks, and naturally infected ticks were found.

Hosts: Pigs, wart hogs, forest hogs, and bush pigs are proven reservoirs. The American javelina is resistant.



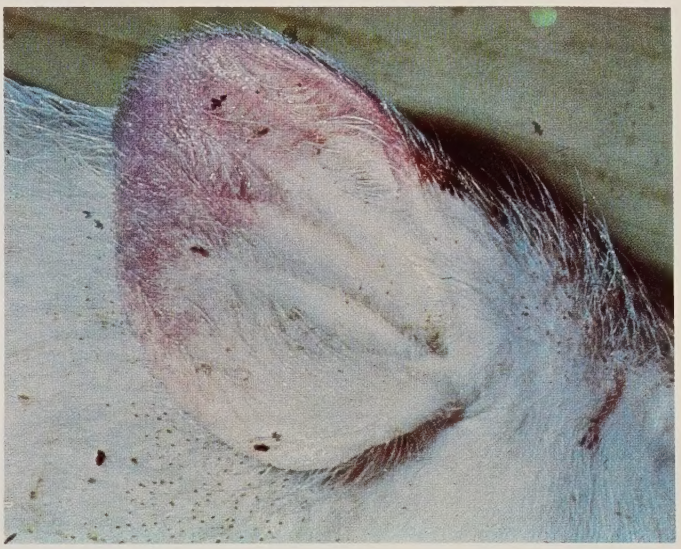
1 Cyanosis of pendant areas of a pig dead of ASF.

* Chronic and low virulente forms of ASF with less well defined signs and much lower mortality may now predominate, even in initial outbreaks.

Clinical Sign: In acute and subacute forms, the incubation period is 5 to 15 days. Fever, depression, lachrymal discharge, cough, diarrhea, dehydration, and death are typical signs; the course of the acute disease is 6 to 12 days. The course may be much longer in the chronic disease!

Gross Lesions: Lesions closely resemble those of hog cholera (HC), except that they may be more severe. Hemorrhages are found on the epicardium and endocardium. Lymph nodes are hemorrhagic. Enlargement of the spleen is frequent in classic ASF. Infarcts are common in HC, but much less so in ASF. Petechial hemorrhages of the kidneys and urinary bladder occasionally are found.

As ASF became enzootic in Spain and Portugal, the signs and lesions in a large proportion of animals lessened in severity and were more similar to those of HC than they had been previously.

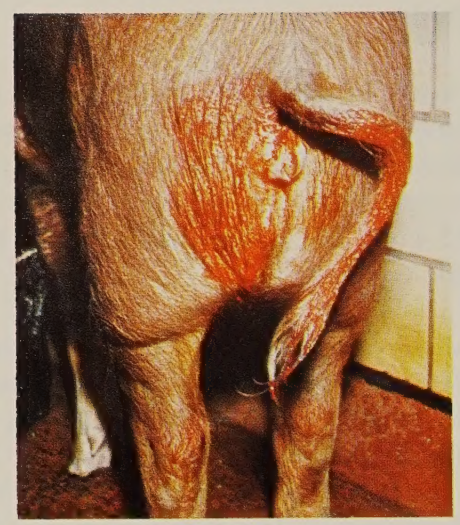


2 Cyanosis of the tip of the ear (ASF).

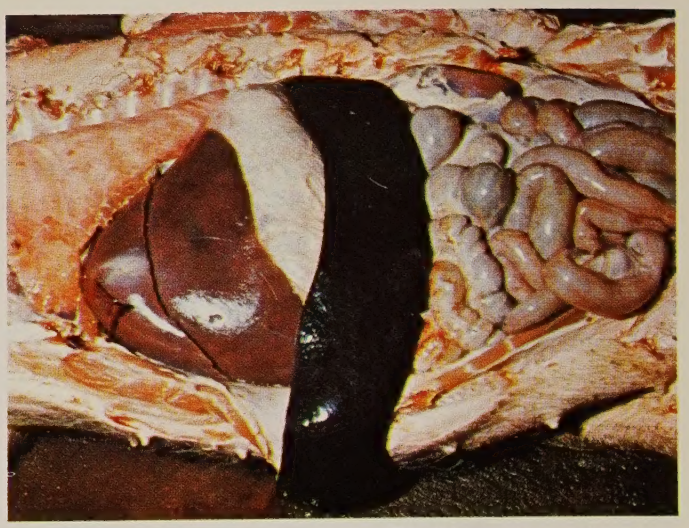
Diagnosis: Disease signs and lesions may or may not be suggestive of ASF. Marked severity of lesions, especially in pigs which were previously vaccinated for HC, may lead to a presumptive diagnosis.

Differential Diagnosis: The disease should be differentiated from HC, erysipelas, and salmonellosis. Appropriate specimens must be taken for all suspect diseases.

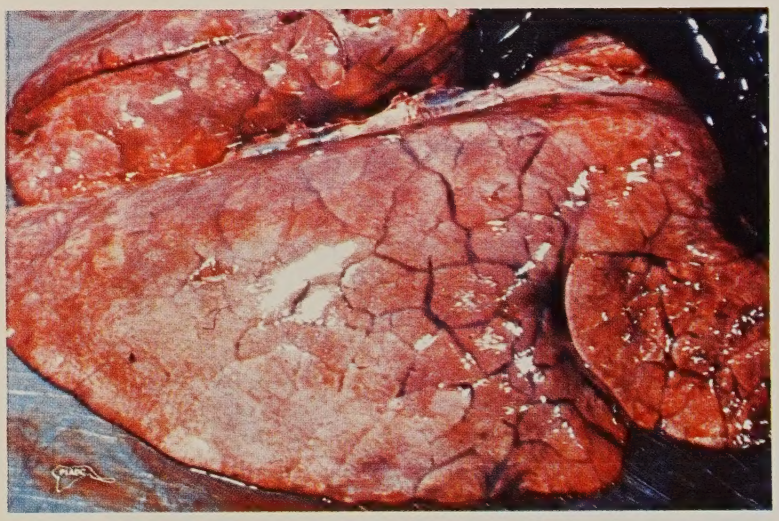
Collection of Specimens for Laboratory Confirmation: For virus isolation, spleen, gastrohepatic, and me-



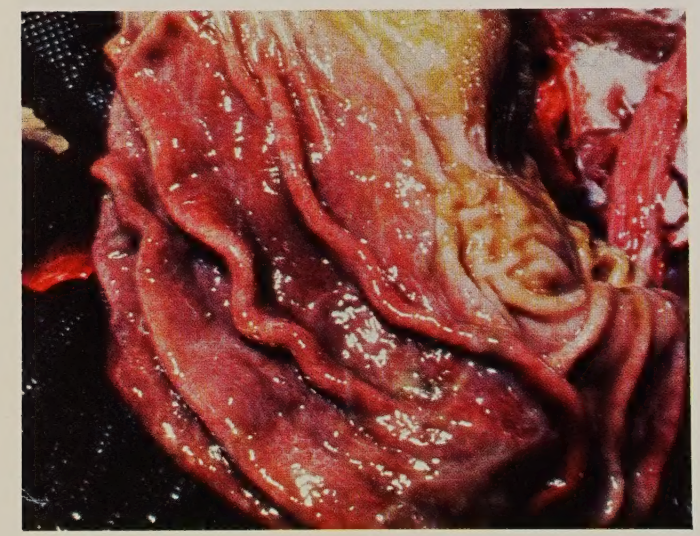
3 Bloody diarrhea (ASF).



6 Typical enlarged friable spleen (Blackberry jam spleen) (acute ASF).



4 Interlobular edema in the lung of a pig dead of acute ASF.



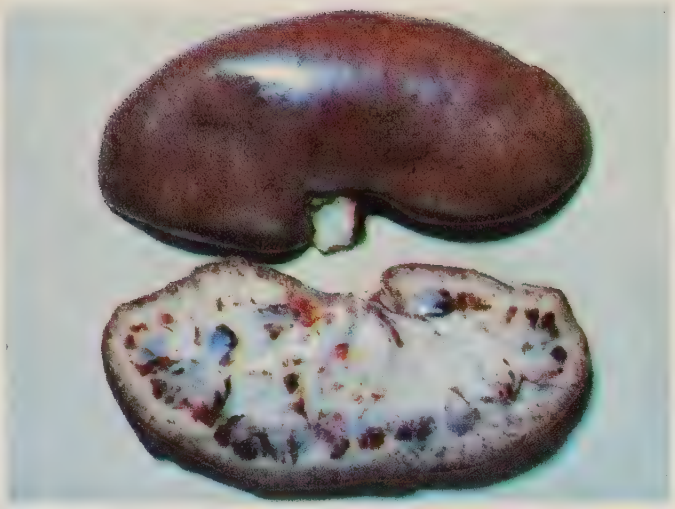
5 Congestion in the fundic portion of the stomach (acute ASF).

senteric lymph modes are the organs of choice since they contain high virus concentrations. They are shipped to the laboratory preferably on dry ice. Where chronic ASF is suspected, serum should be obtained from swine infected longest and shipped frozen.

Laboratory Diagnosis: African Swine Fever can be diagnosed in the laboratory by (1) inoculation of suspect material into cell cultures, immune and susceptible pigs; (2) demonstration of the hemadsorption reaction in pig buffy coat cultures inoculated with blood or spleen suspensions from the suspect pig; and (3) paired sera from the suspect animals which lived the longest may be tested by the AGDP or the immunoelectroosmophoresis (IEOP) and ELISA tests. However, these serologic tests necessitate the use of specially prepared cell culture antigens and known ASF immune serum. The fluorescent antibody (FA) technique for testing liver, spleen, and other frozen tissue sections and smears, (and infected cell cultures) is also a reliable method for detection of ASF.

Recent comparative research on ASF and HC by the Commission of the European Communities (Rabot, 1971) reports the following: "Non-purulent panencephalitis. . . involving both the grey and the white matter is a very important characteristic of HC." This was found in 72 percent and satellitosis in 60 to 100 percent of the HC cases investigated. On the other hand, brain damage in ASF was characterized by cell degeneration, ranging from acute swelling and simple retraction to the severe cellular disease of Nissl; perivascular mononuclear infiltration was generally "not very severe."

7 Spleen from pig infected with ASF at top, contrasted with normal size spleen below.

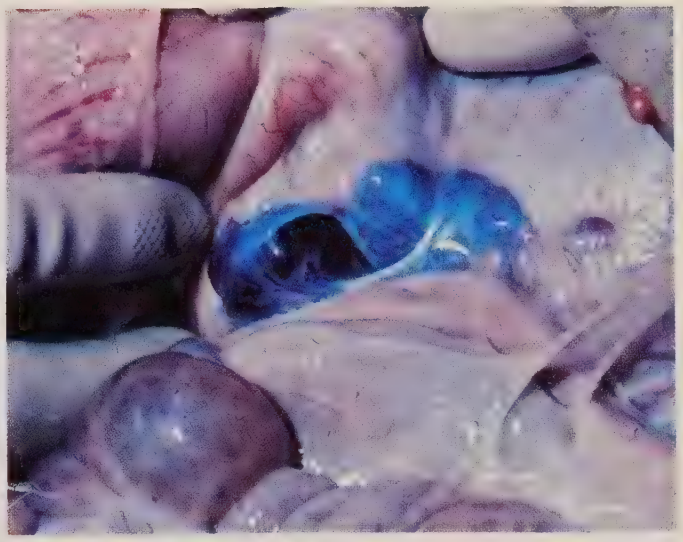


10 Areas of hemorrhage in the pelvis and renal papillae of the kidney in acute ASF.

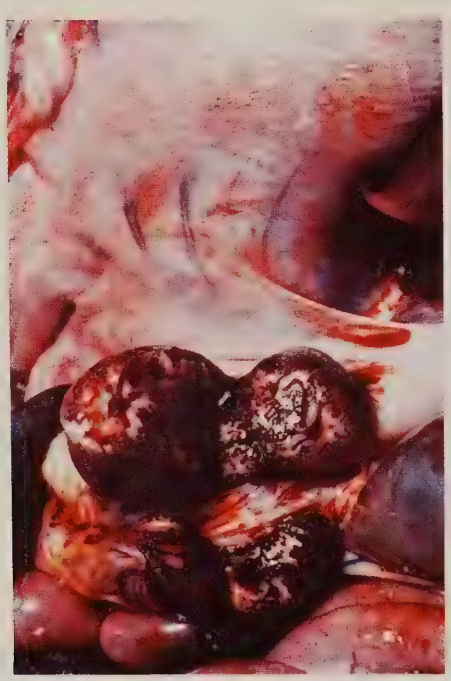
8 Enlarged brown-colored spleen of pig with acute ASF (Brazilian strain).

11 Diffuse hemorrhage in the pelvis of the kidney (acute ASF).

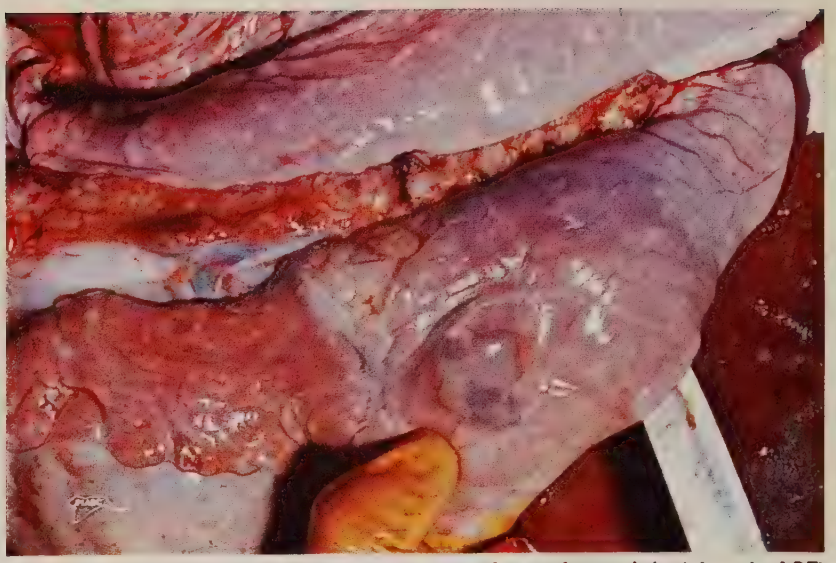
9 Petechial and ecchymotic hemorrhages in the kidney (acute ASF).



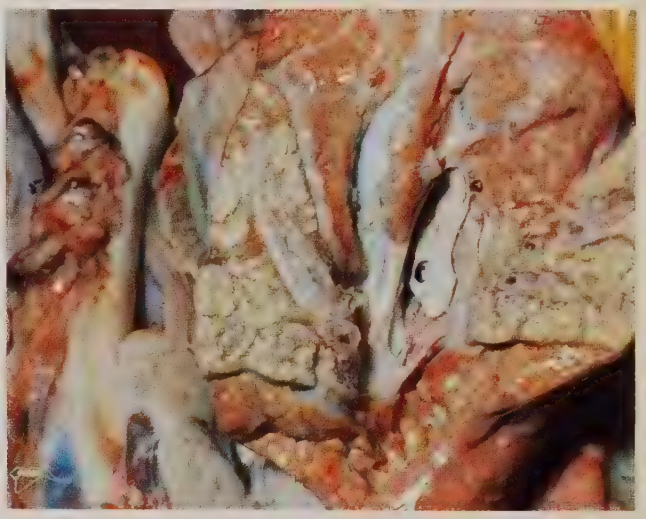
12 Enlarged hemorrhagic hepatogastric lymph nodes which appear as blood clots (acute ASF).



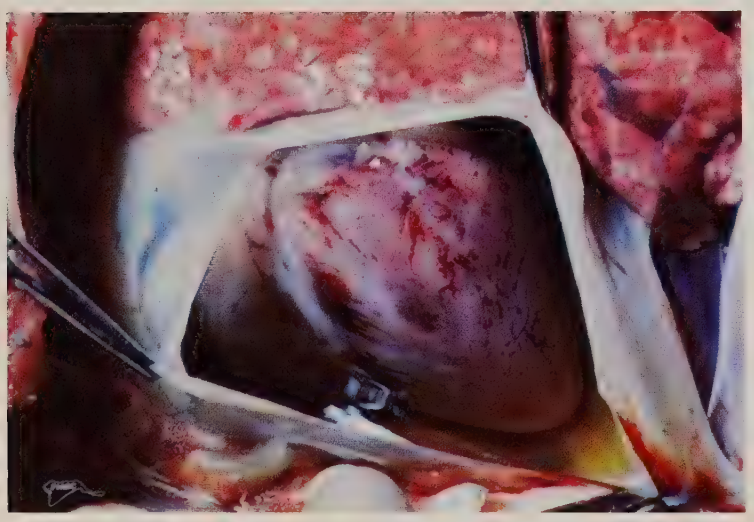
13 Cut surface of a hepatogastric lymph node showing the increase in size and dark red color (acute ASF).



16 Granulomatous lesion in the lung, in the form of a nodule (chronic ASF).

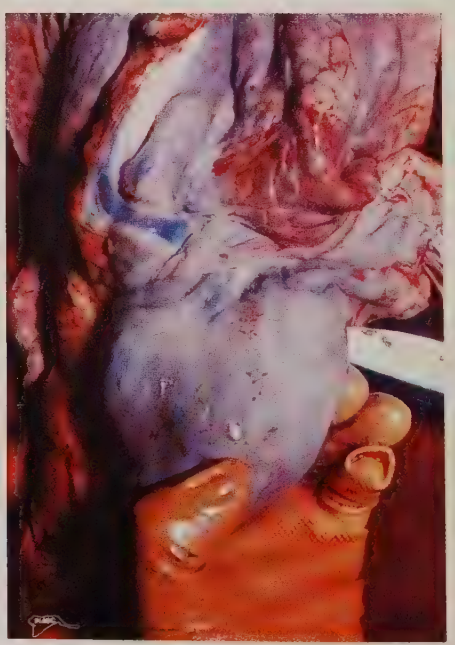


17 Areas of consolidation in the lung (chronic ASF).



14 Pericardial fluid and diffuse hemorrhages in the myocardium (acute ASF).

15 Edema of the gall bladder (acute ASF).



18 Epicardium covered with fibrin and showing some petechial hemorrhages (chronic ASF).

1 Pigs tend to huddle and pile up as if they were cold, when affected with hog cholera (HC).

2 Pigs affected with HC appear weak and staggering and tend to sit like a dog.

Definition: The acute disease is a highly infectious viral septicemia, characterized mainly by generalized hemorrhages.

Etiology: The causal agent is a virus which belong to the Togaviridae family. The agent remains viable at a pH of 5-10 and therefore is not destroyed by post-mortem changes. However, a temperature of 56°C will inactivate it in a few minutes. There is no antigenic variation among the different strains of HC virus. The virus is closely related to the virus which causes bovine viral diarrhea.

Geographic Distribution: Occurs in practically all hog-raising countries. U.S. is now free of HC.

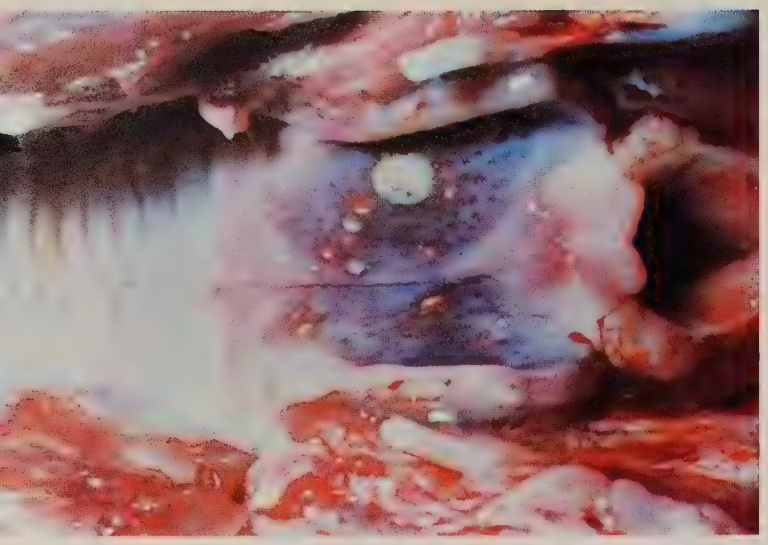
Hosts: The pig is the only animal in which HC is known to occur naturally.

Clinical Signs: Usually appear 5-10 days after infection. At beginning of an outbreak young pigs may die peracutely without clinical signs; however, acute cases are most common. Affected pigs are depressed, do not eat and stand in a drooped attitude with the tail hanging. They are disinclined to move and when forced, do so with a swaying movement of the hind quarters. They tend to lie down and burrow into the bedding, often piled one on top of the other.

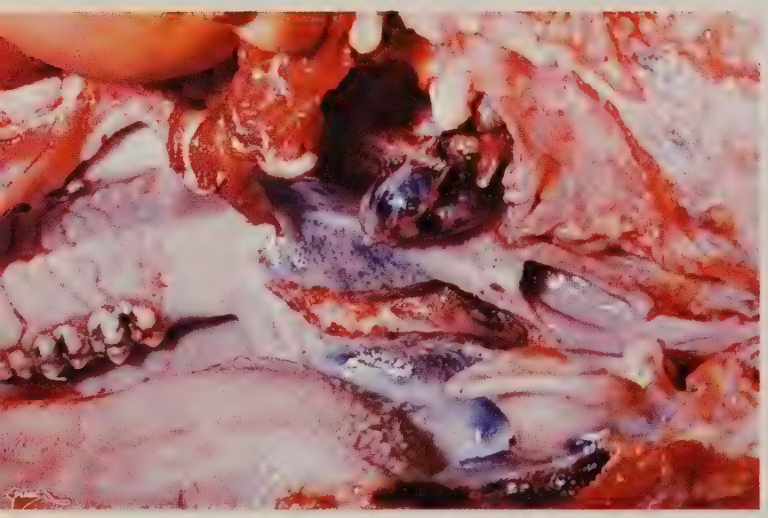
Prior to the appearance of other signs a high temperature is usual (40.5-41,5°C). Other early signs include constipation followed by diarrhea and vomiting. Later a diffuse hyperemia and purplish discoloration of the abdominal skin occurs. Small areas of necrosis

3 Pig showing erythema in different area of the skin (HC).

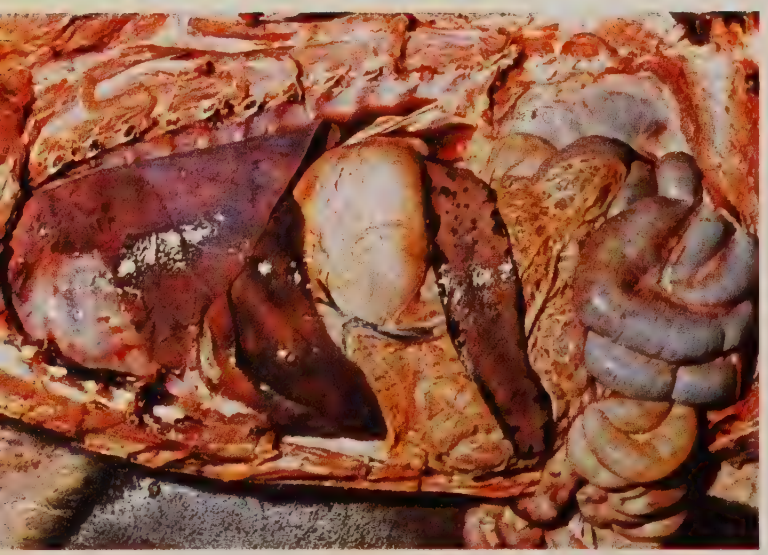
4 Some pigs affected with HC have diarrhea.



5 Pustules and severe congestion on the tonsils (HC).

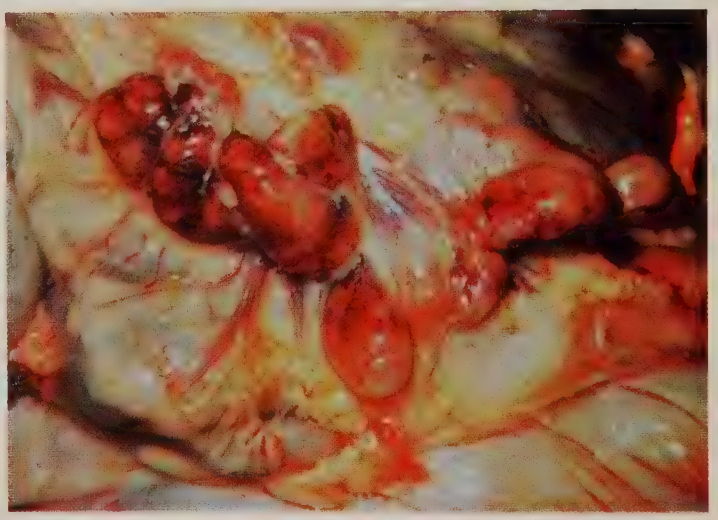


6 Enlarged and hemorrhagic mandibular lymph nodes (HC).

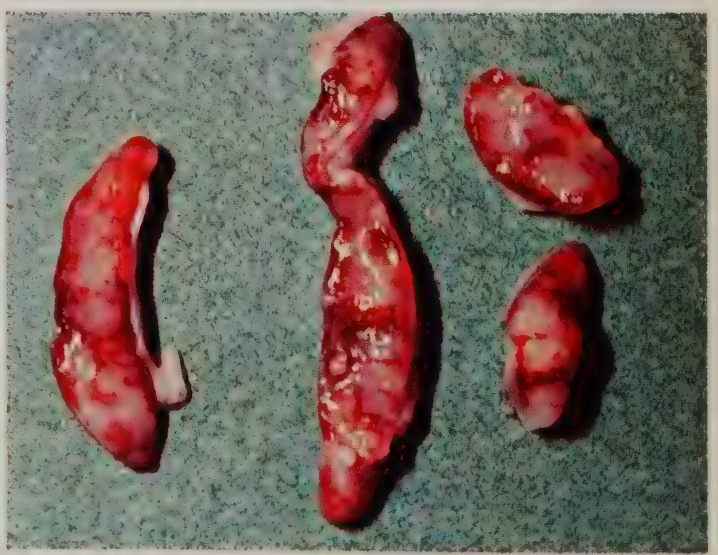


7 Normal-sized spleen with infarcts at the margins (HC).

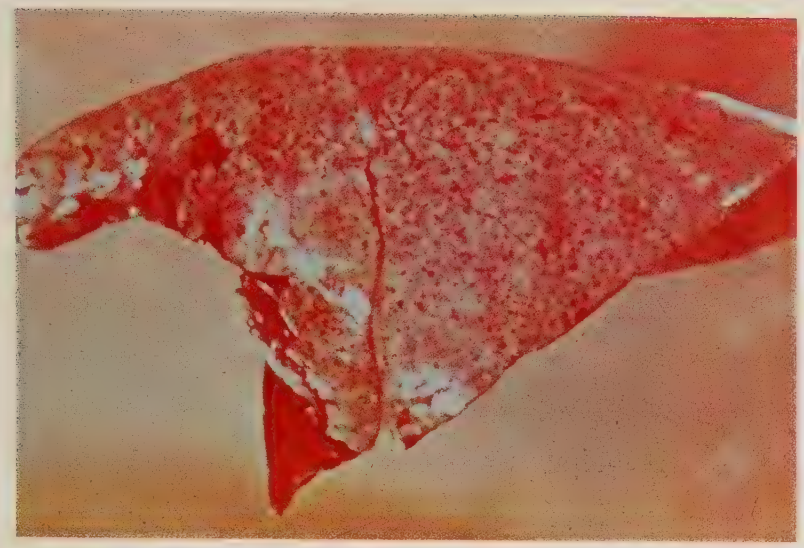
8 Close-up view of marginal infarcts in the spleen (HC).



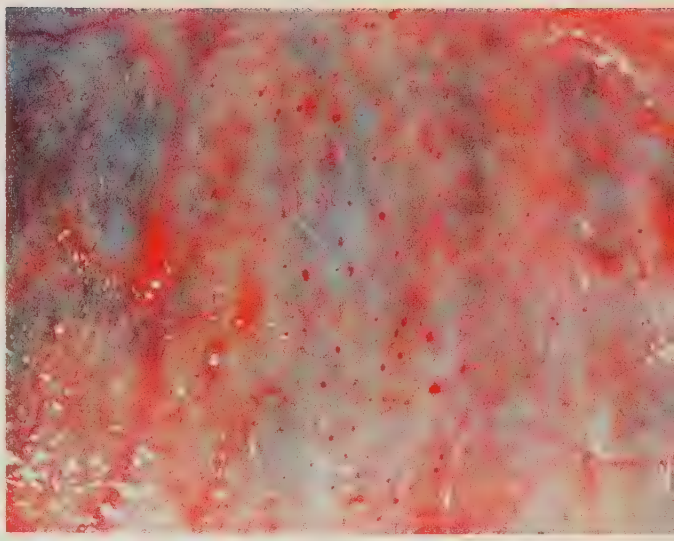
9 Enlarged and hemorrhagic mesenteric lymph nodes (HC).



10 Peripheral hemorrhage in enlarged lymph nodes (HC).



11 Petechial hemorrhages in the lungs (HC).

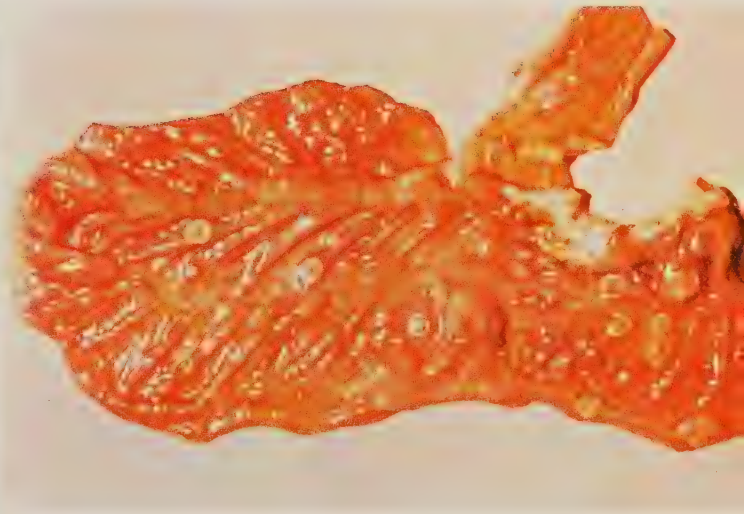


14 Petechial hemorrhages in the mucosa of the urinary bladder (HC).

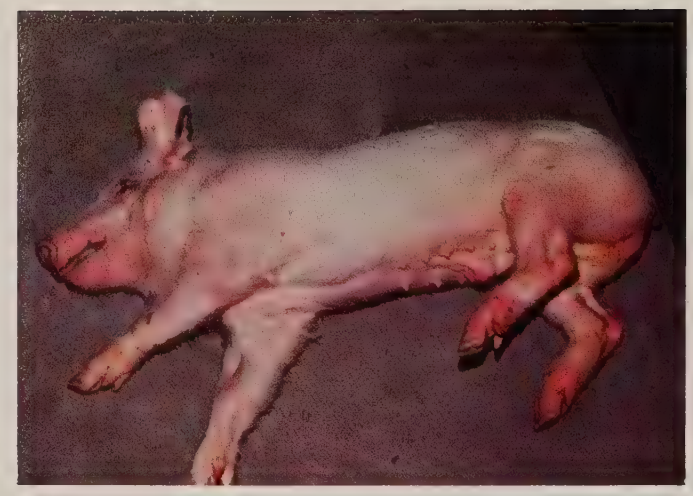
12 Petechial hemorrhages in the kidney (Turkey-egg kidney) (HC).

15 Petechial and ecchymotic hemorrhages in the small intestine (HC).

13 Petechial hemorrhages in the cortex of the kidney (HC).

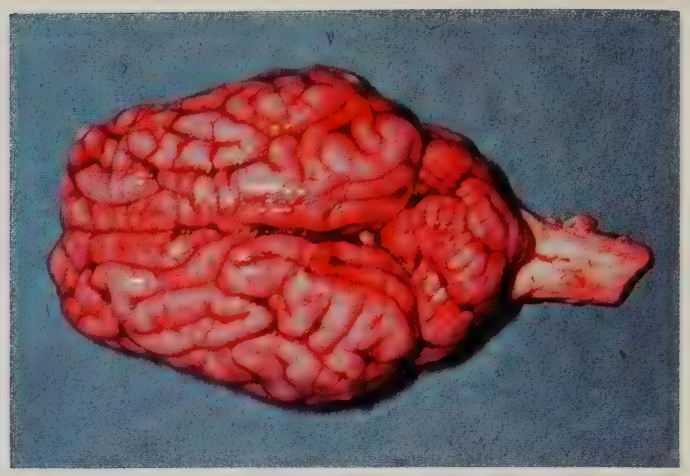


16 Button ulcers in the colon (HC).



17 Running movements, a nervous sign in HC.

18 "Goose stepping" (HC).



19 Venous congestion in a brain (HC).

are sometimes seen on the edges of the ears, on the tail, and lips of the vulva. A degree of conjunctivitis is usual and in some pigs the eyelids are stuck together by dried purulent exudate. Nervous signs are often observed, even in the early stages of the disease, Circling, incoordination, muscle tremors and convulsions are the commonest manifestations. Death can be expected 7-15 days after the commencement of illness.

With the recognition of low virulence strains of virus, less dramatic syndromes have been recognized. A chronic form occurs in field outbreaks, and occasionally after serum-virus simultaneous vaccination. In these cases, the incubation period is longer than normal and there is emaciation and the appearance of characteristic skin lesions including alopecia, dermatitis, blotching of the ears and a terminal, deep purple coloration of the abdominal skin. Infection of pregnant sows may result only in a mild pyrexia, but may be followed by a high incidence of abortion, low litter size, mummifications, stillbirth, and anomalies of piglets. Live born pigs, although carriers, may be weak or clinically normal.

The vaccination of pregnant sows with live HC virus vaccines can cause similar problems or can cause problems of immune tolerance in the newborn pigs, which are born normally, and remain in good condition while the passive immunity acquired from their dams exists, but when this is lost in 20-30 days, these animals can develop viremias that can cause mortalities and also start outbreaks because of reversion to virulent form of the vaccine virus.

Gross Lesions: In peracute cases there may be no gross changes at necropsy. In the common, acute form there are many submucosal and subserosal petechial hemorrhages, which are most noticeable under the capsule of the kidney, near the ileocecal valve, in the cortical sinuses of the lymph nodes, and in the bladder and larynx. Enlargement of the lymph nodes is constant and the spleen and mucosa of the gall baldder may contain marginal infarcts. There is congestion of the liver and bone marrow, and often the lungs. Circular raised button ulcers in the mucosa of the colon are highly suggestive but are now seldom seen. These findings cannot be considered as diagnostic unless accompanied by additional clinical and epidemiological evidence of the disease, since they can occur with other diseases, particularly salmonellosis. In the chronic form of the disease, necrotic ulceration of the mucosa of the large intestine is usual and transverse calcification of the distal portion of the ribs is seen. Secondary pneumonia and enteritis commonly accompany the primary lesions of hog cholera.

Diagnosis: A positive diagnosis of HC is always difficult to make without laboratory confirmation. This is

particularly true of the chronic, less dramatic forms of the disease. A highly infectious, hemmorrhagic, fatal disease of pigs, with a course of 7-15 days in a group of unvaccinated animals should arouse suspicion of hog cholera.

Differential Diagnosis: The major diseases which resemble HC include *salmonellosis,* which is usually accompanied by enteritis and dyspnea, *acute erysipelas,* in which the subserous hemorrhages are likely to be ecchymotic rather than petechial, and *acute pasteureliosis, viral encephalomyelitis* and *salmonellosis,* which produce similar nervous signs. *African swine fever,* apart from its greater severity, is almost impossible to differentiate from hog cholea without serological tests.

Collection of Specimens for Laboratory Confirmation: When hog cholera is suspected, tissues submitted for examination should include blood samples. The brain, and sections of intestine and other internal organs should be preserved in 10% formalin, and untreated pancreas, lymph node, the entire tonsil and large pieces of spleen should be shipped unpreserved in sealed containers under refrigeration.

Laboratory Confirmation: Fluorescent antibody techniques allow rapid detection of antigen in frozen tissue sections or impression smears and in infected cell cultures. The agar gel precipitation test detects antigen in tissues by means of a precipitin formed with immune sera, preferably using pancreas from suspect pigs as the source of the antigen. Antibody can be detected by the fluorescent antibody neutralization test, the tissue culture serum neutralization test, or an indirect enzyme-labelled antibody test which uses enzyme labelled anti-porcine gammaglobulin to detect antigen-antibody combination.

FOOT-AND-MOUTH DISEASE

The four vesicular diseases discussed in this manual (foot and mouth disease, vesicular stomatitis, swine vesicular disease and vesicular exanthema) cannot be differentiated clinically, and the lesions pictured below in cattle can be caused by either foot and mouth disease or vesicular stomatitis, and those in pigs by any one of the four diseases.

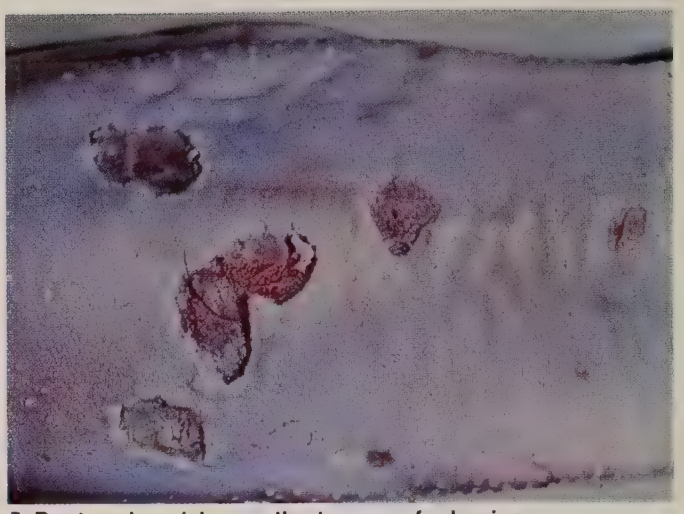
Definition: Foot-and-mouth disease (FMD) is an acute, highly communicable disease existing almost exclusively in cloven-footed animals, domesticated and wild. The disease is characterized by the formation of vesicles and erosions in the mucosa of the mouth and external nares (especially on the snout of pigs) and the skin between and above the hooves of the feet; other areas, including mammary tissue, may be involved.

Etiology: The disease is caused by a virus first isolated in 1897; it is classified with the enteroviruses as a member of the picornaviridae. It has a single-stranded ribonucleic acid core with a protein coat which appears to consist of 32 capsomeres forming a symmetrical icosahedral capsid with a diameter of about 23 nm. There are 7 immunologically and serologically distinct types of virus identified as Types O, A, and C, Southern African Territories (SAT-1, SAT-2, SAT-3), and Asia-1. Within the 7 types at least 61 subtypes have been designated by CF tests.

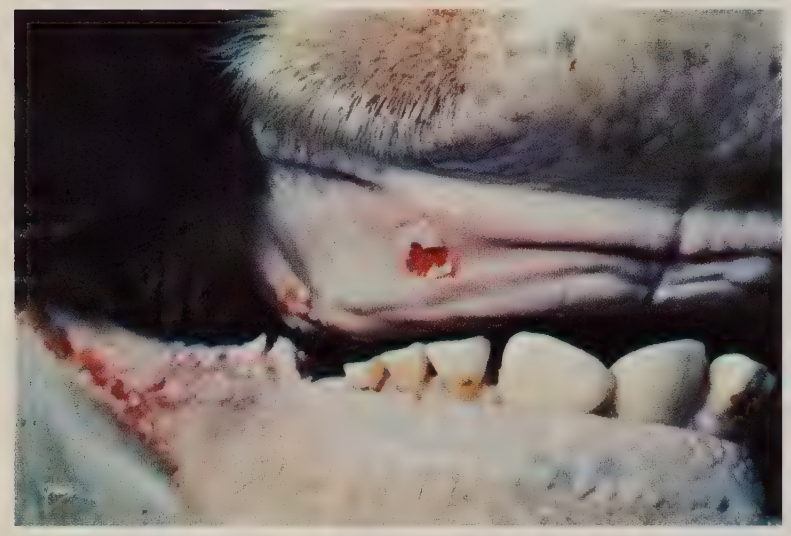
1 Excessive salivation in a bovine with vesicular disease.

Geographical Distribution: FMD occurs in most of the major livestock producing countries of the world, except North America, Central America, Australia, New Zealand, Japan, and Ireland. Several countries in Europe, especially Great Britain and some of the Scandinavian countries are generally free for periods

2 Intact vesicle on the tongue of a bovine.

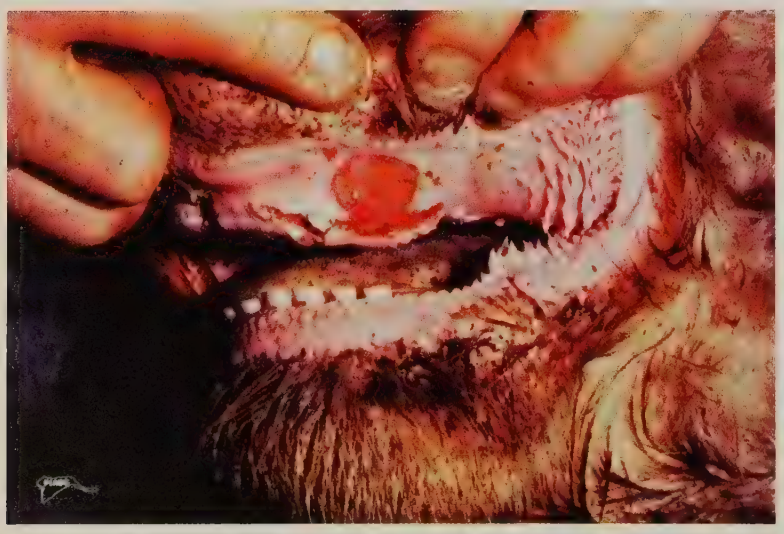


5 Ruptured vesicles on the tongue of a bovine.



3 A recently ruptured vesicle on the mucosa above the dental pad.

6 Extensive area deunuded of epithelium on the tongue of a bovine.



4 Ruptured vesicle on the upper lip and buccal surface of a bovine.

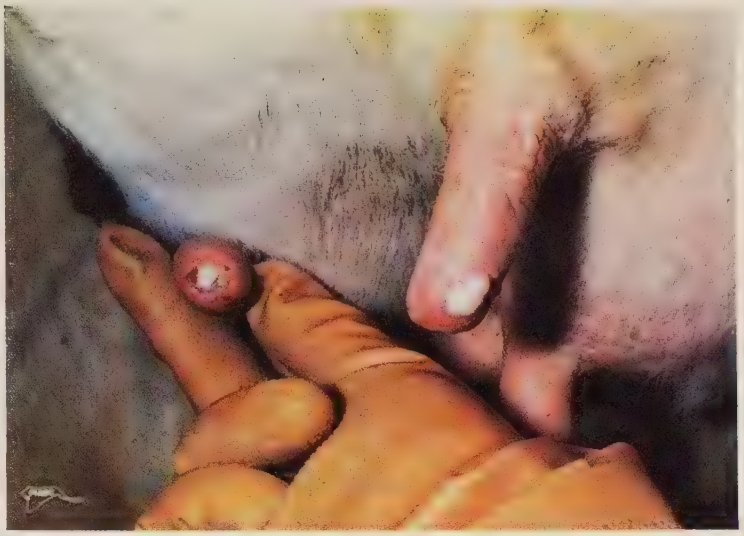
of several years; for example, FMD has not occurred in Great Britain since 1968.

Type Distribution: Types O, A, and C occur in various parts of the world while the African types, SAT-1, SAT-2, and SAT-3, were not found outside Africa until 1962 when an epizootic due to SAT-1 occurred in the Middle East. Asia-1 has been identified from Pakistan, India, Israel, Iran, Iraq, Hong Kong, Thailand, and other Near and Far Eastern countries.

Transmission: The virus is transmitted by contact with infected animals (aerosols primarily), by infected animal products, and by contaminated objects.

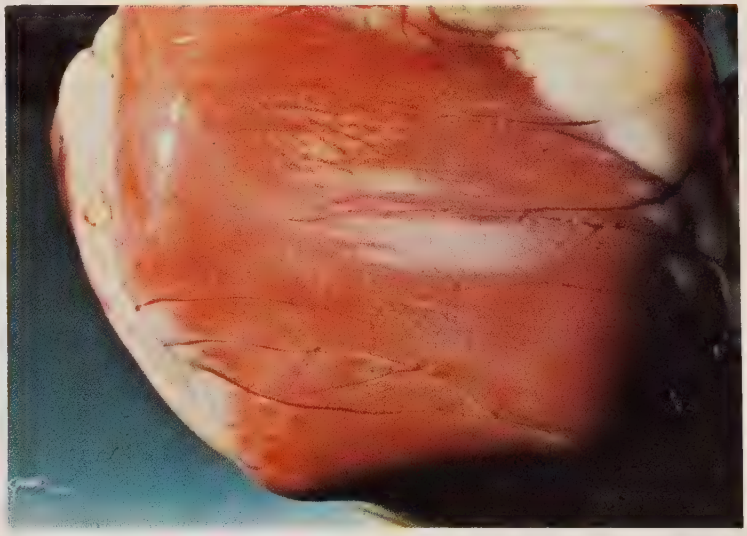
Hosts: All cloven-hooved animals, domestic and wild, are naturally susceptible; pathogenicity for some species is reduced in certain strains. The hedgehog, muskrat, armadillo, and perhaps other wild animals besides the cloven-hooved ones are susceptible; in addition, a wide variety of laboratory animals and cell culture systems can be infected with FMD virus. Man is rarely infected but is capable of transmitting the virus.

Clinical Signs: In cattle, characteristic signs are a moderate pyrexia, lassitude, anorexia, excessive salivation, smacking of the lips, and drooling; these accompany the formation, rupture, and erosion of vesicles of the mouth. When the feet are involved, lame-

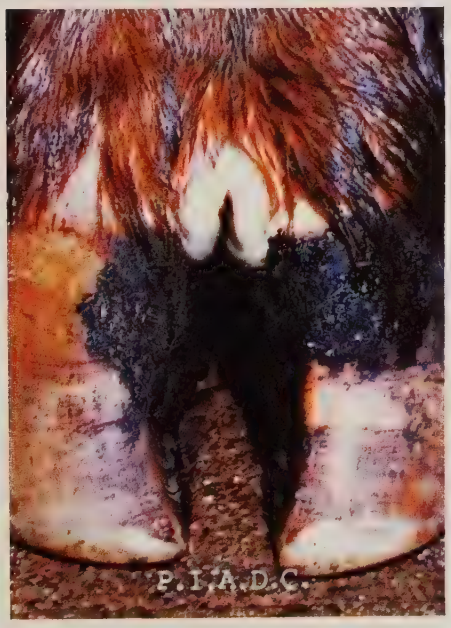


9 Vesicle at the end of the teats in a cow.

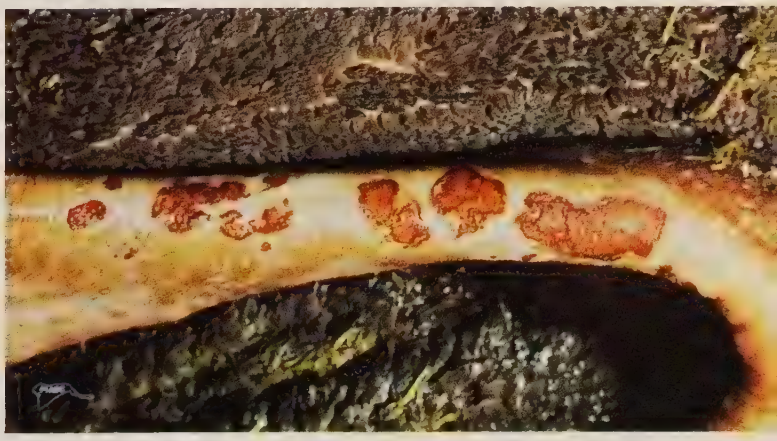
7 Detached epithelium on the tongue of bovine.



10 Necrotic streaks in the myocardium (tiger heart) ocassionally seen in FMD.



8 Vesicle in the interdigital space of a bovine.

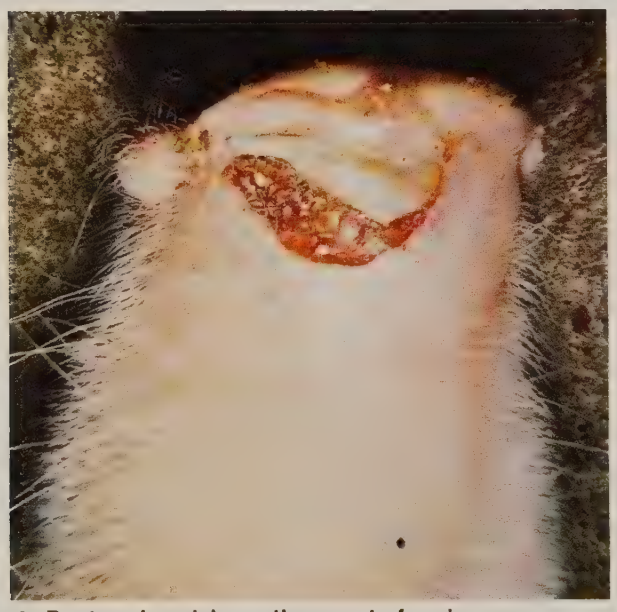


11 Necrosis in the rumen pillars of a calf (FMD).

12 Calf mortality in FMD.

ness is seen. Reduced lactation, mastitis, and abortions are common. Mortality in young animals may be as high as 50 percent but is seldom above 5 percent in adults. Swine show many similar signs; lameness with a changed gait may be quite evident. The incubation period is from 1 to 5 days or longer.

Gross Lesions: Vesicles are *not* pathognomic for FMD alone, since they are also associated with vesicular stomatitis (VS), vesicular exanthema of swine (VES), and swine vesicular disease (SVD). Classical vesicular lesions may not be found; when they occur they usually rupture leaving eroded, hemorrhagic, granular mucosal surfaces of the nose and mouth, as well as the skin, epithelial tissues of the feet and other regions.



13 Ruptured vesicle on the snout of a pig.

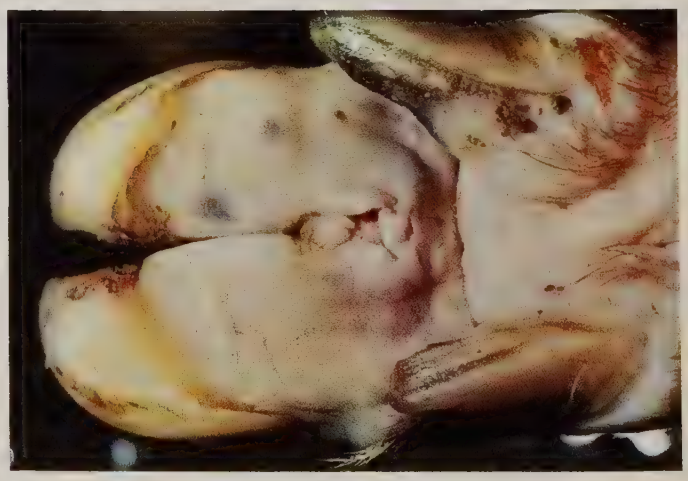
Gastrointestinal lesions may be found at necropsy, particularly of the rumen. In rare cases lesions of the perineum, vulva, or scrotum are seen. Tiger heart (gray, white, or yellowish myocardial lesions) may be seen in calves. In swine and sheep, lesions on the tongue are usually smaller than those of cattle.

Diagnosis: Diagnosis by clinical signs is virtually impossible.

Differential Diagnosis: The inoculation of susceptible horses, swine, and cattle (brought from a region distant from the outbreak) with suspect material may be helpful in differentiating one vesicular disease from another. All three of the species are susceptible to VS; cattle and swine are susceptible to FMD; swine alone respond to SVD and VES. However, laboratory confirmation is necesary.

Collection of Specimens for the Laboratory: Specimens include the following:

Esophageal-pharyngeal fluid obtained with a probang deposited in sterile tissue culture medium containing antibiotic; vesicle fluid collected with aseptic technique in a sterile vial; lesion scrapings placed in tissue culture medium containing antibiotic; paired sera from individual animals or sera from separate

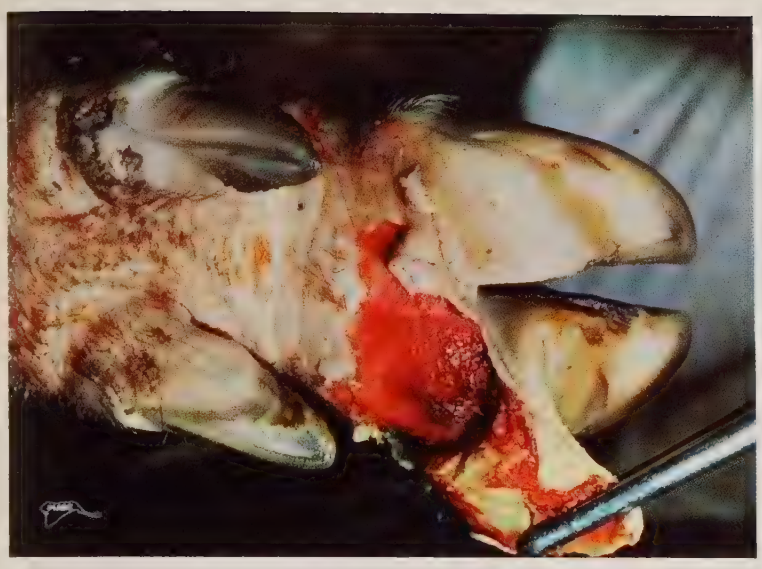


14 Vesicle in the interdigital space of a pig's foot.

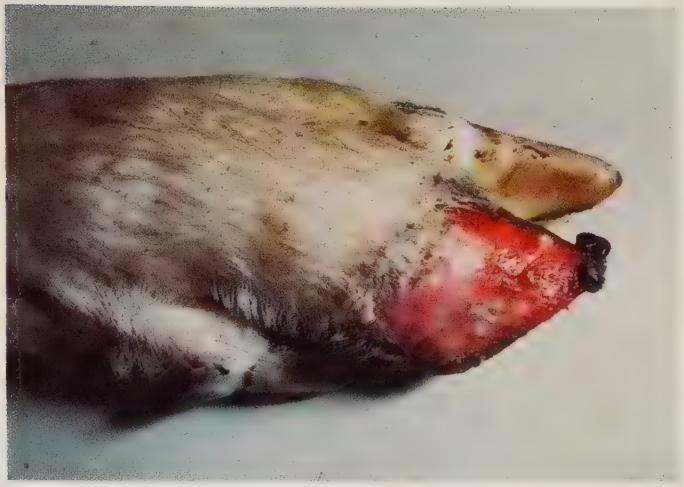
animals taken at early and later stages. All specimens are immediately frozen for shipment (preferably) or placed in glycerol.

Laboratory Confirmation: Laboratory tests for confirmation include:

Complement fixation, the AGDP, virus neutralization, and cross-immunity tests.



15 Detachment of the epithelium, pig's foot.



16 Loss of the horny covering of a pig's toe, following the development of vesicular lesions in FMD.

Control: In countries where the disease is endemic, incidence of the disease is controlled by vaccination programs. In an increasing number of countries vaccination is mandatory; in others it is voluntary. In countries that are generally free of the infection, the disease is eradicated by slaughter followed by disinfection of the premises. The animal carcasses are generally destroyed by burning or burial. While costly, this method is considered to be the most effective way to deal with an outbreak.

VESICULAR STOMATITIS

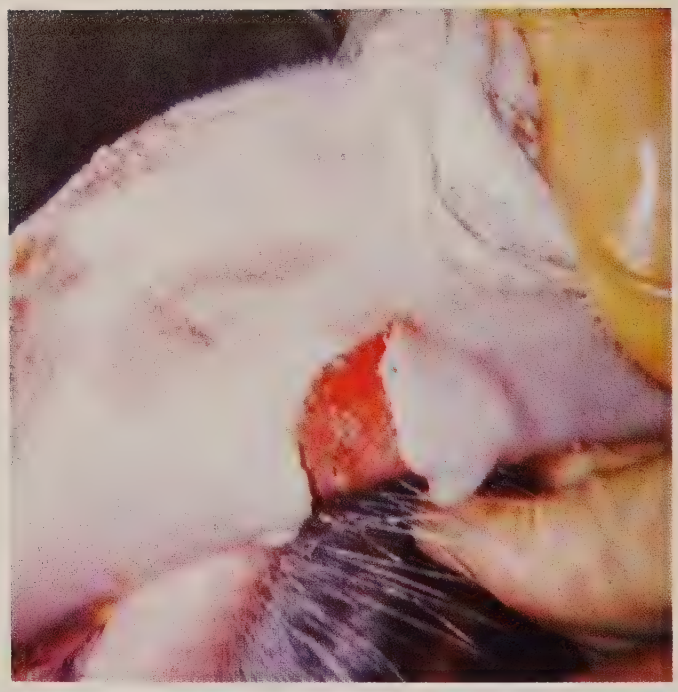
Definition: Vesicular stomatitis is a viral disease of horses, cattle and swine. Macules, vesicles and erosions appear successively in the mucous membranes of the mouth or on the skin of the teats or the foot. Mortality or serious sequelas are rare.

17 In some cases almost the entire epithelial surface of the tongue is detached.

Etiology: The causal agent is a virus belonging to the Rhadboviridae family, of the vesiculovirus genus. It is sensitive to changes in pH, especially acid. Serologically, the VS virus has two distinct serotypes, New Jersey and Indiana, with the latter being made up of three subtypes, Indiana I, Indiana II (Cocal) and Indiana III (Alagoas). Even though the NJ and Indiana serotypes are serologically and immunologically distinct, the infections are clinically indistinguishable.

Geographic Distribution: Clinical vesicular stomatitis occurs mainly in the Western Hemisphere. There are enzootic areas of the NJ type and the Indiana subtype I in the U.S., México, Central América, Panamá, Venezuela, Colombia, Ecuador and Perú. Only the NJ type has been found in Bolivia and Canadá.

Transmission: The ecology of the vesicular stomatitis virus is still not very well understood. There are many questions as to where and how the virus is maintained in nature, how it is transmitted from one animal to another, and how it is introduced into herds free of infection. The Indiana and New Jersey viruses may have different cycles. It has been found that the infection caused by the Indiana virus in enzootic areas is frequent in wild arboreal or semi-arboreal animals, and that the agent has been isolated from mites *(Gigantolaelaps)*, tropical sand flies *(Phlebotomus)* and from mosquitoes (Aedes and Culex). *Phlebotomus* can transmit the infection transovarially to its progeny



18 Ruptured vesicle on bovine tongue.

and to susceptible animals by biting them. Serologic conversion has been observed in sentinel monkeys placed in individual cages in endemic forest areas in Panamá. These facts plus the fact that the disease occurs most frequently when arthropods are most abundant suggest that, at least for the Indiana virus, there may be a trasmission cycle between wild animals and arthropods. However, several objections to this hypothesis have been raised. Viremia induced in various animals is insufficient to infect biting arthropods. Moreover, the capricious distribution of the disease during outbreaks, which sometimes leaves contiguous farms unaffected, is difficult to explain. Also, there have been epizootics during which it has not been possible to isolate the virus from arthropods. Other hypotheses suggest that the virus is in the soil or pasture and that the animals become infected by inoculation, either through the skin or oral mucosa, or that the reservoir of the virus could be a plant or insect, with vertebrates being only accidental hosts. The replication of the NJ virus in arthropods after feeding on a natural host has so far not been confirmed.

Man contracts the infection by contact with domestic animals, either through the nasopharyngeal route, through abrasions in the skin, or by aerosols. The direct sources of infection may be saliva, the exudate or epithelium of open vesicles, or the virus itself when handled in laboratories.

Hosts: VS has been isolated from infected horses, cattle and hogs. There is ample serologic evidence of natural infection in wild animals. In Panamá, antibodies were found for the Indiana type 1 in arboreal and semi-arboreal species of animals, and for the NJ type in bats, carnivores and some rodents. Infections in man have been found mainly in laboratory workers and persons in enzootic areas exposed to domestic animals.

Clinical Signs: The incubation period is from 2 to 4 days. The symptomatology is similar to that of foot and mouth disease, with which it can be easily confused. The disease is characterized by a short period of fever and the appearance of papules and vesicles in the mouth, on the teats, in interdigital areas, and on the coronary bands. Profuse salivation is often the most prominent sign. The location of the vesicles may vary depending on the outbreaks; in some they may be predominantly located in the mouth and in others on the teats. Foot lesions occur in some outbreaks and not in others, being most frequent in swine. Affected animals generally recuperate in a period of 1 week. The most common complications are secondary bacterial infections, mycosis, and mastitis. The disease can cause appreciable economic losses, primarily by affecting dairy cattle and swine.

Gross Lesions: Limited to the epithelial tissues of the mouth, teats and feet.

Diagnosis: Vesicular stomatitis in cattle and swine is indistinguishable from clinical cases of foot and mouth disease in these animals. Swine are also affected by swine vesicular disease and vesicular exanthema which also produce clinical signs which cannot be differentiated from those of VS or FMD. A presumptive diagnosis of vesicular stomatitis in horses can be made on clinical evidence since there are no other viral or bacterial diseases that produce a similar set of signs and lesions in these animals. Caustic substances or photosensitivity may produce similar lesions, but the history should permit a differentiation. Rapid laboratory diagnosis is very important in domestic animals to distinguish VS from FMD. The most useful test is the complement-fixation test, using the epithelium of the vesicles as antigen. The virus can easily be isolated from vesicle material (epithelium or fluid) in cell cultures or by inoculation of mice.

Differential Diagnosis: (See discussion in section on FMD).

Collection of Specimens for the Laboratory: Epithelial tissue covering the vesicles in the mouth, or on the feet

or teats should be collected and placed in buffered glycerol or frozen for shipment. If available, vesicular fluid should be collected aseptically in a sterile vial and frozen. Esophageal-pharyngeal fluid obtained with a probang should be deposited in sterile tissue culture medium containing an antibiotic and also frozen.

Paired acute and convalescent serum samples can be used for CF or SN tests to show a rise in VS antibodies.

Laboratory Confirmation: Laboratory tests for confirmation include CF and SN tests.

SWINE VESICULAR DISEASE

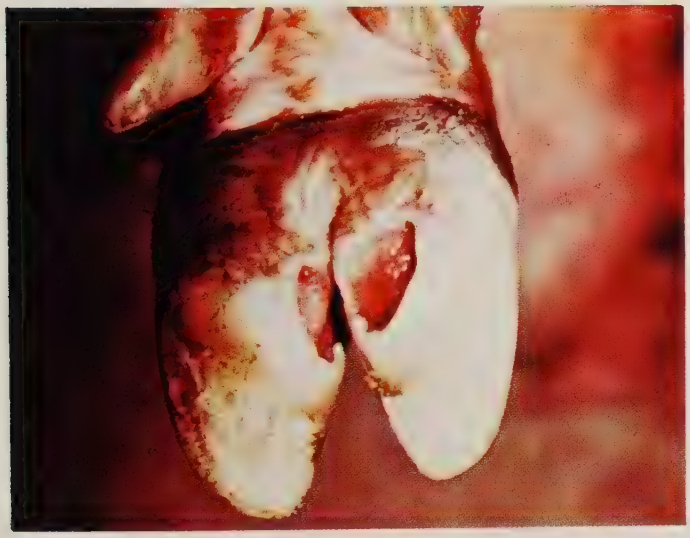
Definition: Swine vesicular disease (SVD) is a contagious viral disease of swine indistinguishable in the field from foot-and-mouth disease (FMD), vesicular stomatitis (VS) and vesicular exanthema of swine (VES). It is a relatively new disease in that its first appearance was described in 1966.

Etiology: The infectious agent of SVD is a porcine enterovirus in the Picornaviridae. The virus is a roughly spherical particle of 150 S sedimentation rate, a density of 1.34 grams per ml in cesium chloride, and a diameter of about 280A. It is acid and ether stabile with single-stranded RNA, stabilized at 50 C by 1 M $MgCl_2$.

The virus of SVD (SVDV) is serologically and biologically closely related to the human entereovirus, Coxsackie B-5.

Geographical Distribution: In 1966 a disease indistinguishable from FMD was observed in Lombardy, Italy. Failure to confirm an initial diagnosis of FMD resulted in laboratory studies which identified it as an enterovirus. In 1970 pigs in Hong Kong were vaccinated for FMD with an inactivated virus; in April of 1971 a vesicular condition, first diagnosed as FMD, was observed in the vaccinated pigs. Further studies revealed that this was the same enterovirus previously described in Italy. In 1972 FMD was diagnosed in pigs in Staffordshire, England and slaughter of swine and cattle started. Five days later, laboratory studies showed that this was not FMD but the same enterovirus previously encountered in Italy and Hong Kong. The new disease, now termed SVD, was soon identified in France, Poland, Austria and again in Italy. In late 1973, Germany and Switzerland were added. In November 1973, the disease was reported in Japan; by 1974 it has spread to 15 different foci. The disease appears to be unchecked in both Europe and Asia.

Transmission: The appearance of SVD in Great Britain and other countries of Europe and also in Japan was found to be related to the recent importation of pork products or pigs from countries known or thought to have SVD in swine. In addition to the ingestion of



19 Ruptured vesicles on a pig's foot.

20 Unruptured vesicle on the dorsal surface of the snout of a pig.

virus in garbage, animals within herds become infected by contact with pigs shedding SVDV in their excretions, particularly the feces. Due to the viremia in SVD, all tissues contain virus and can serve as a source of infection.

The skin of pigs has been found to be much more susceptible to infection by SVD than by FMD. It is believed that viral contamination of minor wounds and scratches is a means of transmission of SVD. Pigs carried in trucks which had previously transported SVD-infected animals were infected even though the trucks had been decontaminated. Restocking proved difficult on some British farms due to reinfection. The virus of SVD is stable under a variety of environmental condition for many months. For example, SVDV could be isolated from the surface and gut of earthworms collected from the soil above the buried carcasses of infected pigs.

Hosts: Swine and man are the only knows species to be naturally infected. Newborn mice are readily infected by intracerebral or intraperitoneal inoculation of SVDV but 7-day-old mice are refractory.

Several laboratory workers who had contact with SVD-infected pigs or SVDV developed a variety of illnesses traceable to infection with SVDV but not the related Coxsackie B human enterovirus.

Clinical Signs: Swine vesicular disease is usually first detected by the sudden appearance of lameness in several animals in a herd. On soft ground this may be overlooked. Where the animals are on hard surfaces they may be observed to limp, stand with arched back, or refuse to move even in the presence of food. These signs have maximal expression in the larger and heavier animals. The temperature is usually elevated 2 to 4 degrees C. Lesions usually appear along the cornonary bands and interdigital spaces of one or more feet. Vesicles appear and rupture resulting in ulcerous skin lesions extending to the metacarpus and metatarsus with loosening of the sole pad. Vesicles and resulting ulcerations may also be found on the snout, epithelium of the buccal cavity, the tongue and teats.

The incubation period of SVD is from 2 to 4 days for the appearance of vesicles at the inoculation sites and from 5 to 6 days for generalization of infection with vesicle formation at secondary sites. Recovery from SVD is usually rapid with pigs returning to normal within 3 weeks. Morbidity is moderate and mortality usually low. However, in the experimental infection of a sow with newborn pigs, there were both high morbidity and mortality in the pigs.

Gross Lesions: The gross and microscopic appearance of vesicular lesions of SVD are essentially the same as that described for FMD. No gross lesions other than those related to vesiculation have been found.

Diagnosis: There are no clinical signs that will help to differentiate SVD from FMD, VES or VS. In every instance regarding the initial outbreaks it is well to remember that they were diagnosed as FMD. The absence of a vesicular disease in cattle in contact with diseased pigs might be suggestive of SVD, but it should be remembered that FMD viral strains have been isolated from pigs which had very low infectivity for cattle.

Any vesicular condition should be reported and action initiated to obtain a laboratory diagnosis.

Differential Diagnosis: See the chapter on FMD. Presence of a vesicular condition in cattle would tend to eliminate SVD (although there could be the possibility of multiple infections in some regions). Vesicular disease in horses might suggest VS. Differential diagnosis requires the use of laboratory tests.

Collection of Specimens for Laboratory Confirmation: See the chapter on FMD. Vesicular fluids are collected, if available, separately from unruptured vesicles and frozen. Vesicular lesion tissues: collect about 5 grams in phosphate buffered glycerin (volumetric measurement of 5 cc of liquid may serve as a guide). Vesicular lesion materials may also be frozen. Ten ml of whole blood for virus isolation should be collected during the febrile period and frozen. Ten ml of serum should be obtained from animals in the acute and convalescent stages of the disease. Submit frozen or refrigerated.

Fecal samples from animals with and without lesions (for virus isolation) may be submitted frozen.

Laboratory Confirmation: Swine vesicular disease can be differentiated from FMD, VS and VES by a variety of laboratory tests such as CF, virus neutralization, differential growth in cell cultures and measurement of physical and biochemical parameters. The CF and virus neutralization tests are the most specific, and of these, the CF test is the most rapid. Antiserum against the different strains of SVDV for use in the CF test can

be prepared by immunizing guinea pigs with repeated inoculations of infected fluids harvested from cell cultures or brains taken from infected newborn mice. These sera are used in a differential diagnostic CF test which also includes antisera against different types and strains of FMD, VS and VES. The test antigen usually consists of a suspension of vesicular lesion material collected from the diseased animals.

Diagnosis by use of virus neutralization can be done with the same sera used in the CF test or with sera collected from animals which recovered from the different vesicular diseases. Portions of the suspension of vesicular lesion material are mixed with each of the different sera and these mixtures inoculated into cell cultures prepared from cells susceptible to the viral infection. Diagnosis is based upon an absence of cytopathic effect (CPE) in those cultures in which the antiserum is of the same type as the test sample. Virus identification requires about 3 hours by CF and 2 to 4 days by virus neutralization.

Other laboratory methods include inoculation of a variety of cell cultures; SVDV will grow only in swine kidney cultures, while FMDV will grow in bovine kidney as well as swine kidney cell cultures. The virions of FMDV are rapidly destroyed at a pH below 6.5, while those of SVDV remain intact. If a viral agent from vesicular material is isolated in cell culture and then treated at pH 5, examination by electron microscopy will reveal particles if the agent is SVDV but none if the agent is FMDV.

VESICULAR EXANTHEMA

Definition: An acute, febrile, contagious viral disease of swine, characterized by the formation of vesicles on certain parts of the body.

Etiology: Caused by a virus currently classified as a calcivirus.

Geographic Distribution: The naturally occurring disease was reported only in the United States between the years 1932 and 1955. Since that time the disease in swine has not been diagnosed anywhere in the world and it is believed that virus has been eradicated.

Hosts: Vesicular exanthema virus affects only swine. A virus isolated from pennipeds (San Miguel Sea Lion Virus) in 1972 is very similar to VESV and produces a diseases clinically indistinguishable from vesicular exanthema.

Transmission: Vesicular exanthema is known to be spread by at least 2 methods, namely direct contact and the feeding of raw garbage.

Clinical Signs: Are indistinguishable from those seen in swine affected with foot and mouth disease, vesicular stomatitis or swine vesicular disease.

Gross Lesions: Vesicle formation is the only know lesion directly attributable to the infection.

Diagnosis and Laboratory Confirmation: (See sections on FMD, VS and SVD).

21 Unruptured vesicles on the snout of a pig.

22 Pig walking on his knees because of the pain due to vesicular lesions on the feet.

BOVINE PAPULAR STOMATITIS

Definition: A widely distributed virus disease which produces papular and occasionally erosive lesions on the muzzle and buccal mucous membranes of young cattle.

Etiology: Caused by a member of the Poxviridae family which is characterized by high resistance to environmental conditions such as dessication. There is evidence that the viruses of BPS and pseudocowpox are identical.

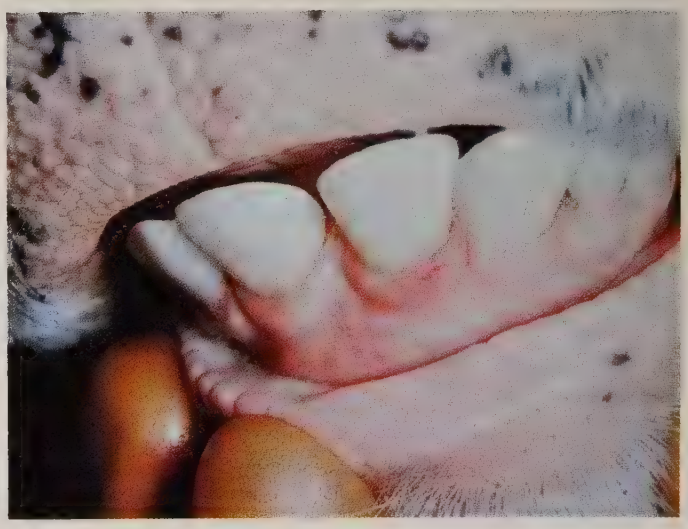
Geographic Distribution: The disease has been reported in the U.S., Africa, Australia, New Zealand, Canada, Great Britain and Europe.

Transmission: Papular stomatitis is seen in bucket-fed as well as nursing calves. While nursing infected cattle may be a major mode of transmission, the disease probably also spreads on feeding utensils. Since it is seen in young animals from 2 weeks up to 2 years of age, various modes of transmission may exist. Insect transmission may also be a possibility. The virus may enter through abrasions in the mucosa.

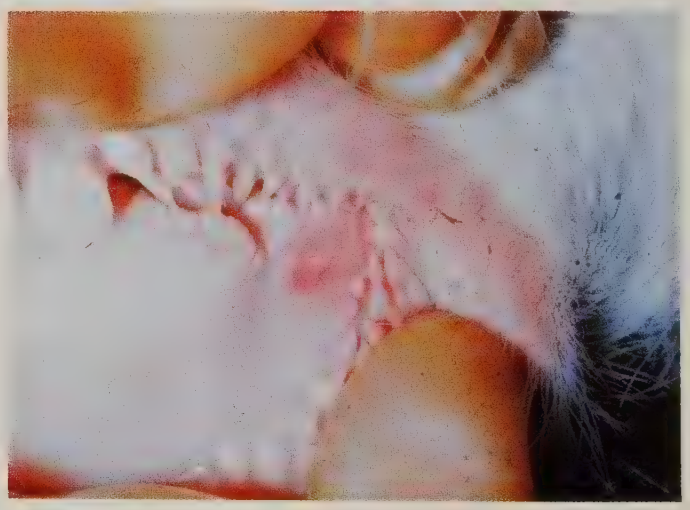
Hosts: Primarily a diseas of calves, the virus can affect man, causing skin lesions.

Clinical Signs: There may be transient anorexia or a slight fever, but in most instances the disease goes unnoticed, unless a careful examination of the mouth is made. Lesions are usually confined to the muzzle, the nostrils and the buccal mucosa. Lesions commence as small papules, which become dark red in color, develop a roughening of the surface and expand peripherally, so that the lesions are always round or nearly so. As the lesion expands the periphery becomes reddened and the center depressed, grey brown in color and rough on the surface, and eventually covered with necrotic tissue, or on external lesions, by a scab. Individual lesions heal quickly, sometimes in as short a time as 4-7 days, but evidence of healed lesions, in the form of circular areas of dark pink mucosa, usually surrounded by a slightly paler raised zone, may persist for weeks. There may be successive crops of lesions in one animal over a period of months.

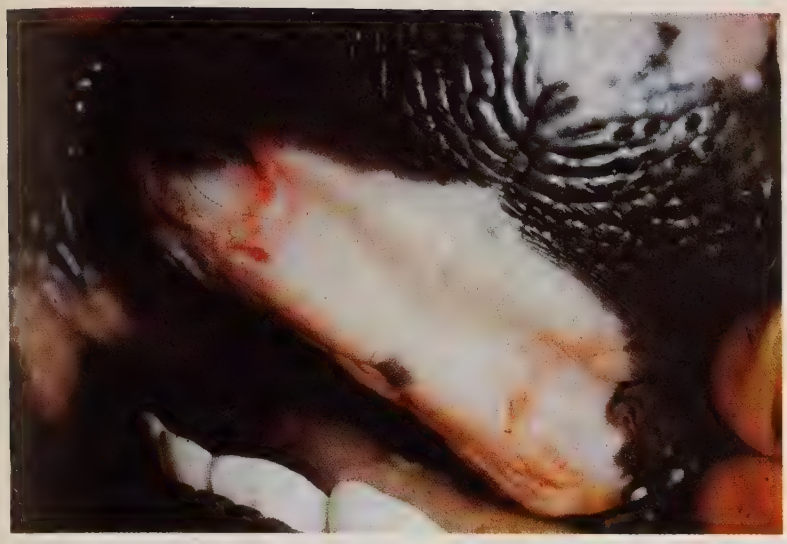
1 Lesion on the muzzle of a bovine with BPS.



2 Gum lesions (BPS).



3 Lesion at the commissure of the lips (BPS).



4 Gum lesions (BPS).

5 Lesions on the hard palate and gums (BPS).

Gross Lesions: The vast majority of infected animals survive without serious harm and are therefore not subjected to necropsy. When papular stomatitis lesions are found at necropsy, they are usually incidental findings.

Diagnosis: Primarily calves 1-20 months of age, and usually less than 6 months, are affected. Patients usually show no systemic disturbance, but have raised papular or flat brown lesions with irregular edges on the oral mucous membranes and the muzzle.

Differential Diagnosis: Includes pseudo-cowpox, vesicular stomatitis, foot and mouth disease, bovine viral diarrhea and rinderpest. Bovine papular stomatitis resembles endemic erosive stomatitis of cattle reported in Africa, proliferative stomatitis, muzzle disease and ulcerative stomatitis.

Collection of specimens for laboratory diagnosis: The disease can usually be diagnosed clinically by the nature and location of the lesions. If there is any possibility that the lesions may be vesicular, specimens should be collected as described in the sections on foot and mouth disease or vesicular stomatitis.

RINDERPEST

Definition: Rinderpest (RP) or cattle plague is an acute, highly contagious virus disease, primarily of cattle, secondarily of sheep, goats, and wild ruminants. Pigs of European and North American origin, when exposed to rinderpest, may develop an inapparent infection with a mild transient fever (although they may transmit virulent virus to cattle). The American javelina and indigenous swine of the Far East are highly susceptible.

Etiology: Rinderpest virus strains are immunologically uniform, but they may vary in virulence. The virus which belongs to the family Paramyxoviridae, is about 300 nm in size and immunologically related to measles and distemper viruses. It is destroyed by strong acids and alkalies.

Geographical Distribution: The disease is enzootic in Asia and Africa but not in Europe or the Americas. The last epizootic in Europe was in Belgium in 1920.

Transmission: Transmission is by contact with infected animals or indirectly with their secretions, excretions, and fomites. The virus appears in the blood and secretions before the appearance of signs. For this reason, the infection may be easily introduced inadvertently to slaughterhouses and stockyards. Animals that recover develop solid immunity and a high antibody titer; they are not known to be carriers.

Hosts: Hosts are chiefly cattle, buffaloes, deer, camels, sheep, goats, and occasionally swine.

1 Excessive salivation follows appearance of oral lesions in RP.

2 RP causes a high morality.

3 Diarrhea is one of the principal signs of RP.

4 Before dying from RP, cattle frequently assume the classical "milk fever" position.

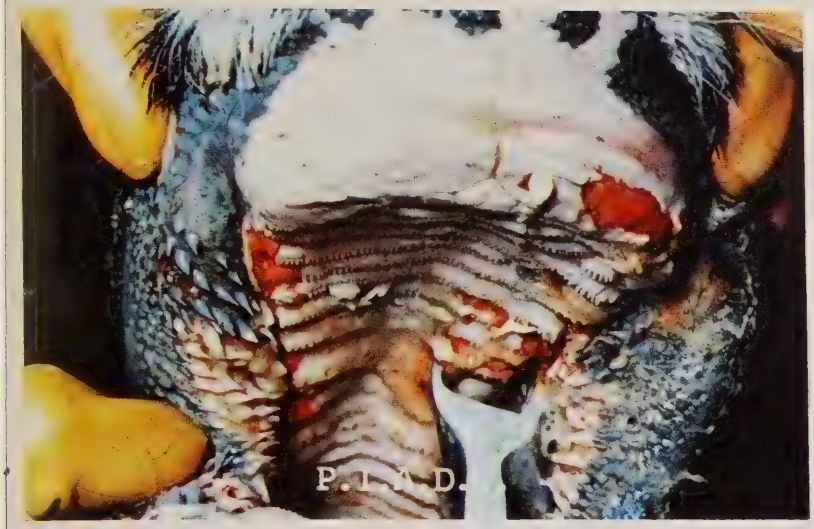
Clinical Sings: The incubation period is ordinarily 3 to 10 days, although where the disease is enzootic it may be longer; the incubation period of the experimental disease may be as short as 40 hours.

The major clinical signs are high fever, nasal discharge, erosions of the buccal mucous membranes, constipation followed by diarrhea, dehydration, rough and soiled hair coat, and death in 7 to 12 days.

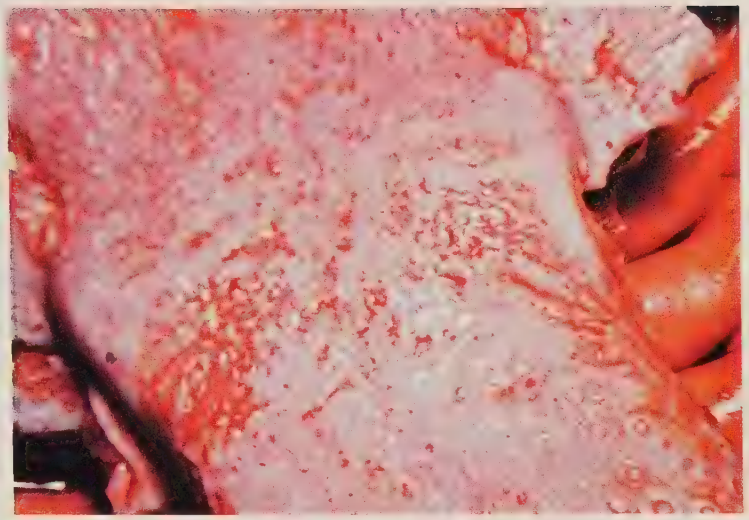
Gross Lesions: Lesions include punched-out-like erosions on the inner surfaces of the lower lip, the gums, ventral surface of the tongue and the soft palate. Upon necropsy, the lymph nodes are edematous. Peyer's patches are acutely inflamed, eroded, severely hemorrhagic, and necrotic. The mucosa of the abomasum is hemorrhagic. There is often edema, hemorrhage and erosions of the mucosa of the cecum, the cecocolic junction and the rectum. The mucosal surface of the last portion of the large intestine usually shows zebra stripe markings.

Diagnosis: The history, signs and lesions are valuable in reaching a diagnosis. However, because of the similarity of these features to those of other diseases, discussed later, a confirmatory laboratory diagnosis is necessary.

Differential Diagnosis: Diseases that resemble rinderpest clinically are acute mucosal diseases, bovine malignant catarrhal fever, acute coccidiosis, and foot-and-mouth disease. Inoculation of animals with these disease agents may not result in mortality (ex-

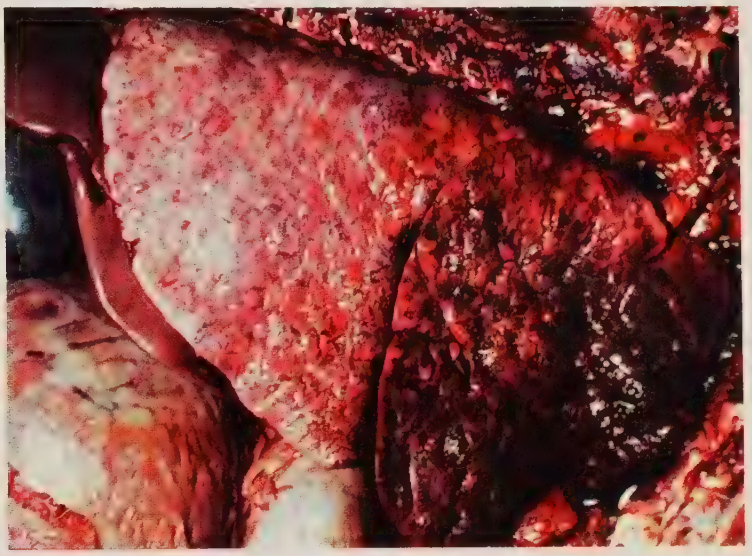


5 Erosions produced by RP on the dental pad and the hard palate resemble FMD lesions.

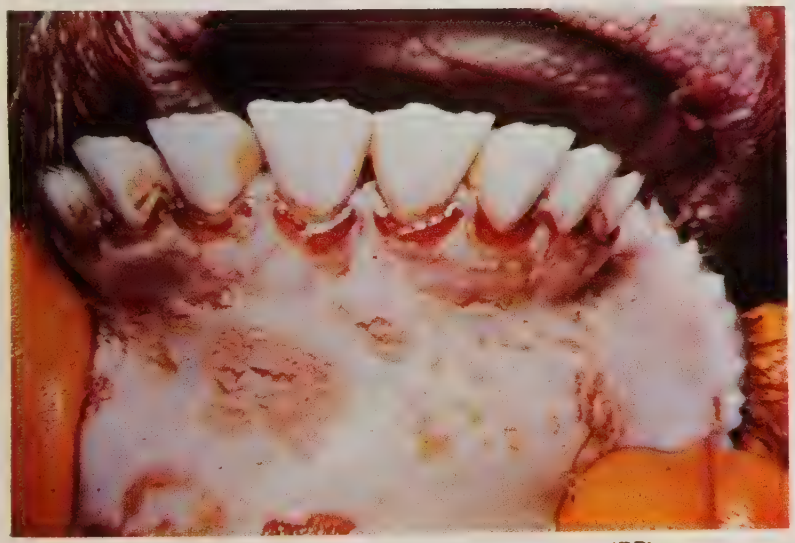


8 Erosions at the base of the tongue (RP).

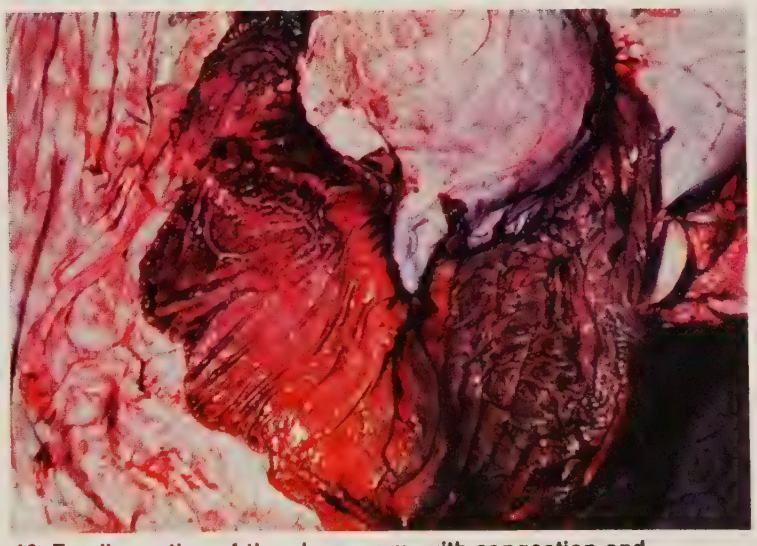
6 Erosions on the gums caused by RP.



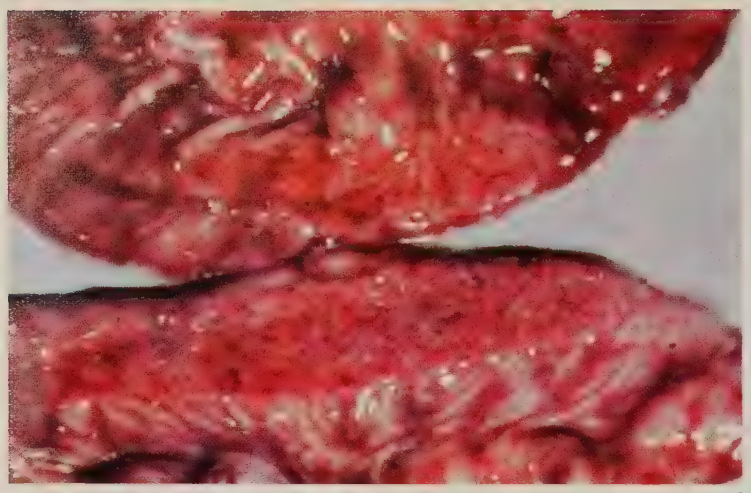
9 Severe congestion of the anterior lobes of the lungs (RP).



7 Lesions on the buccal mucosa and gums (RP).

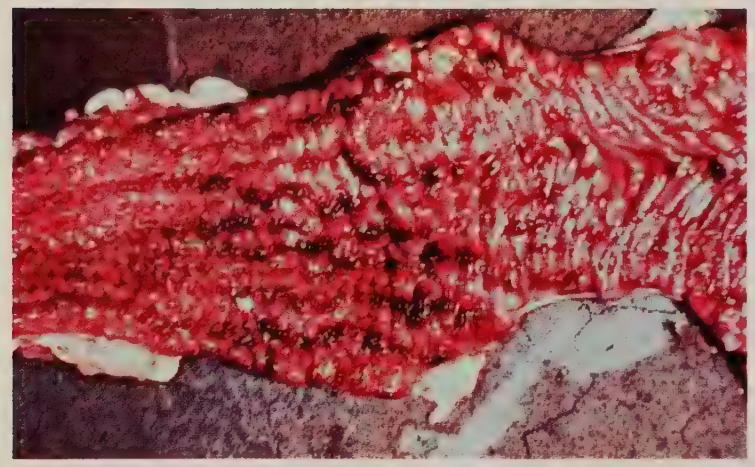


10 Fundic portion of the abomasum, with congestion and hemorrhage (RP).

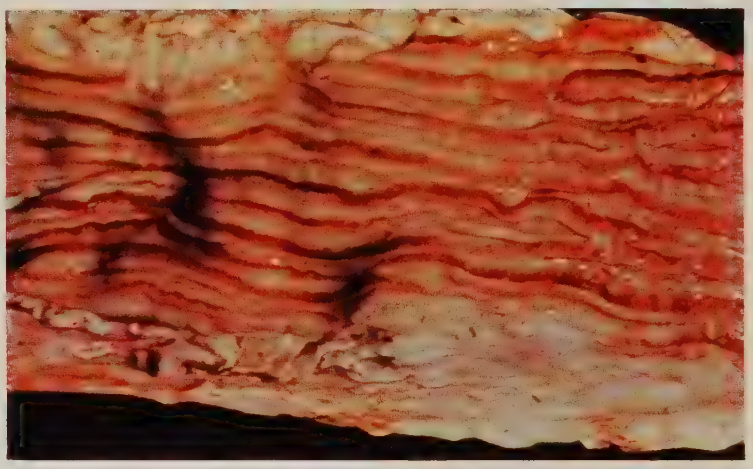


11 The mucosal surfaces of the Peyer's patches are enlarged and congested in RP.

13 Close-up of an area of the small intesine with hemorrhage and erosions (RP).



12 Severe congestion and hemorrhage in the intestine (RP).



14 "Zebra-striping" in the distal colon and rectum (RP).

cept for MCF). Since some strains of rinderpest are of low virulence, these other diseases may present difficulties in differential diagnosis.

Collection of Specimens for Laboratory Confirmation: For virus isolation, heparinized blood, mesenteric lymph nodes, and spleen are collected early in the acute phase of the disease. One portion of the heparinized blood should be shipped refrigerated; other specimens should be received frozen at the laboratory. Serum should also be collected from animals which have been ill for the longest period of time during the outbreak.

15 Enlarged and edematous lymph node (RP).

For histopathology, specimens of tonsils, liver, spleen, kidney, and portions of intestines showing lesions should be collected in 15 percent neutral buffered formalin.

Laboratory Confirmation: Attempts to isolate the virus are carried out in tissue culture or animals. Extracts from lymph nodes of infected animals may be used in the complement fixation (CF) or agar gel diffusion precipitation (AGDP) test as antigens against RP rabbit hyperimmune serum. Virus neutralization tests in cell cultures may be carried out with the sera of animals that were sick long enough to develop antibodies. A definitive diagnosis may be obtained by cross-protection tests using immune and susceptible cattle.

MALIGNANT CATARRHAL FEVER (AFRICAN)

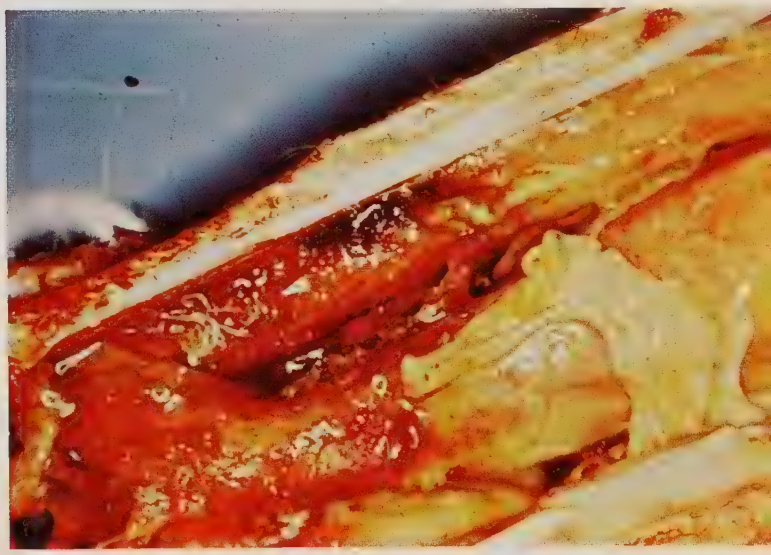
Definition: Malignant Catarrhal Fever (MCF) of Africa, also known as snotsiekte, is an acute generalized disease of cattle and buffaloes characterized by high fever, profuse nasal discharge, severe hyperemia, diffuse necrosis of oral and nasal mucosae, leukopenia, ophthalmia, corneal opacity and enlargement of lymph nodes. Four syndromes are recognized: the peracute, intestinal, head and eye and mild. The natural disease is usually of the head and eye form with low morbidity and high case fatality rates.

1 Corneal opacity and crusted muzzle (MCF).

2 Mucous nasal discharge and crusted muzzle (MCF).

Etiology: The etiologic agent of wildebeest-associated MCF in Africa is a herpesvirus with a capsid about 100 nm and an envelope about 140-220 nm in size. The virulent virus may be isolated from any tissue of the sick animal. Highest titers are found in virus from the buffy coat, lymph nodes and other tissues of the reticuloendothelial (RE) system. It is thought that a similar agent may be the cause of MCF outside Africa since the disease in other continents resembles that seen in Africa. However, no other proven disease agent for MCF has been isolated and the African form has been found elsewhere but only in zoo animals.

Geographical Distribution: The disease syndrome is world-wide and occurs sporadically. A severe epizootic in cattle occurred in Colorado during the winter of 1971-1972. A sheep-associated form was reported in Europe in 1798, Switzerland in 1832, the USA in 1920 and Canada in 1924. Wildebeest-associated MCF was known to South Africans in the early half of the 19th century. The herpesvirus agent was isolated in Kenya.



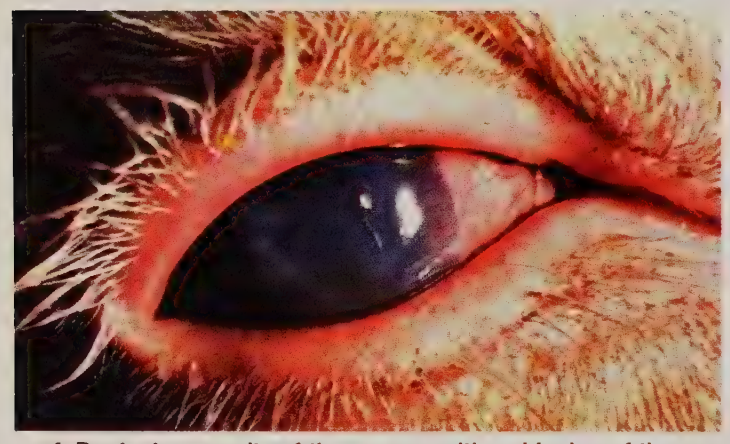
3 Hemorrhage and necrosis of the mucosa of the turbinates (MCF).

Transmission: The disease is transmitted from the natural reservoirs to cattle, the alien host. Wildebeest are natural reservoirs in Africa and sheep are thought to be reservoirs elsewhere. In both sheep and the wildebeest, transmission occurs when cattle are grazed with these animals at or following parturition. Close contact between donor and recipient are regarded as essential. Studies conducted at the Plum Island Ani-

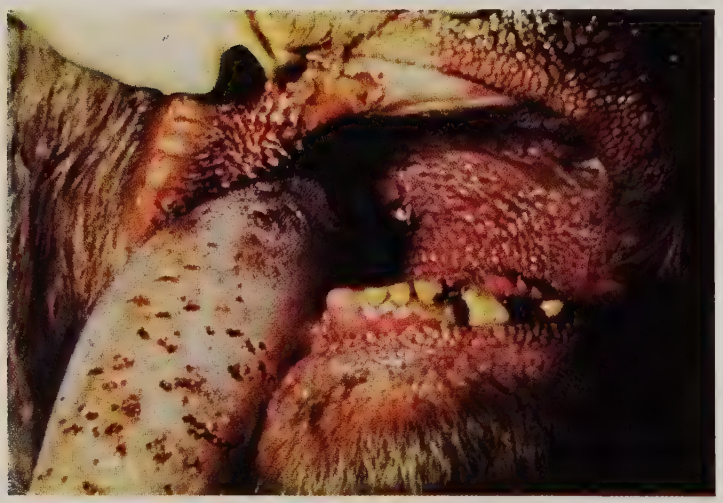
mal Disease Center and in Africa, independently, have shown that nasal discharges of infected cattle carry the African MCF virus. This finding gives a partial explanation as to how the disease is transmitted in nature and indicates that under certain conditions cattle-to-cattle transmission may occur.

Hosts: The blue wildebeest *(Connochaetes taurinus)* and black wildebeest *(C. gnu)* are two known natural hosts of African MCF. They have an inapparent infection. Cattle. in which the disease appears, are secondary hosts. Elsewhere, it is believed that sheep and cattle are the natural and secondary hosts, respectively.

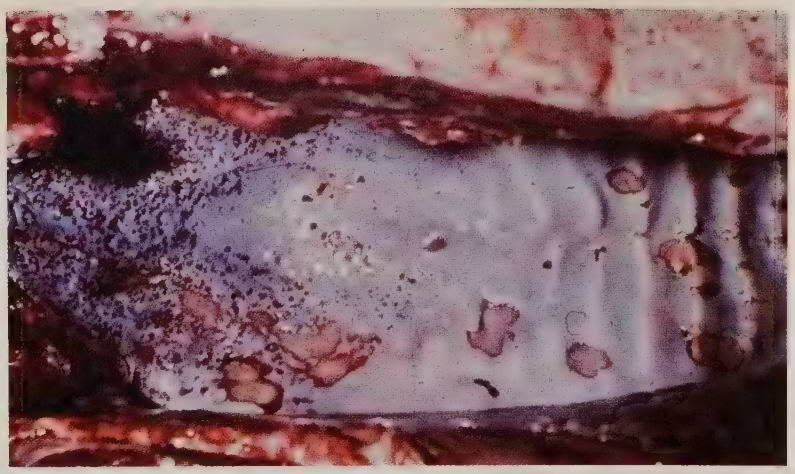
Clinical Signs: The clinical picture of MCF is arbitrarily divided into four forms: the peracute, intestinal, head and eye and mild. There is considerable overlap observed in the syndrome which can be quite variable and the diagnosis elusive.



4 Beginning opacity of the cornea, with reddening of the eyelids and conjuntivitis (MCF).



5 Erosions on the tongue (MCF).



6 Erosions on the hard palate (MCF).

(1) *Peracute Form:* Severe inflammation of the oral and nasal mucosa, and hemorrhagic gastroenteritis are observed. The course of this form is 1-3 days.

(2) *Intestinal Form:* This form is characterized by pyrexia, diarrhea and severe hyperemia of the oral and nasal mucosa. Nasal and ocular discharge as well as enlargement of lymph nodes are common features. The course of this form is 4-9 days.

(3) *Head and Eye Form:* This is the typical clinical syndrome of MCF. The first evidence of infection is pyrexia which is often heralded 2-7 days later by nasal and ocular discharges. Bilateral nasal discharge begins as serous and soon becomes mucoid, mucopurulent and later purulent. Encrustation is common in late stages and causes partial or complete blockage of nostrils resulting in dyspnea. At this stage, the sick animal breathes through its mouth and usually shows drooling of saliva.

The oral mucosa exhibits intense hyperemia and diffuse superficial necrosis. Because the basal layer of the epithelium is rarely involved, the necrotic lesions are designated as erosions rather than ulcers. In the live animals, these lesions have a pink or red color due to exposure of the underlying capillary bed. They are found on the lips, gums, hard and soft palate and the mucosa of the cheeks. The sharp pointed bucal papillae are often involved and the tips slough leaving characteristic reddened, blunted papillae. Petechiae are occasionally present. These changes cause severe pain and the animal objects to the examination of its mouth.

Changes in the eye include lacrimation that becomes purulent in late stages. Ophthalmia, prominent scleral veins and swollen eyelids are common features.

Corneal opacity starts at the periphery and progresses towards the center resulting in either partial or complete blindness. Corneal opacity is usually bilateral but occasionally one eye is affected more severly than the other. Photophobia is usually associated with corneal opacity. An animal exhibiting this sign closes its eyes most of the time and points its head away from the source of light.

Pyrexia is a common sign of the disease and is often biphasic. The temperature is usually high, 104-107 F, and remains high until shortly before death at which time it is subnormal.

Increased thirst starts in early stages of the disease and continues until shortly before death. Anorexia is observed in the late stages of MCF. Constipation is a common feature of the head and eye form but terminal diarrhea is occasionally observed.

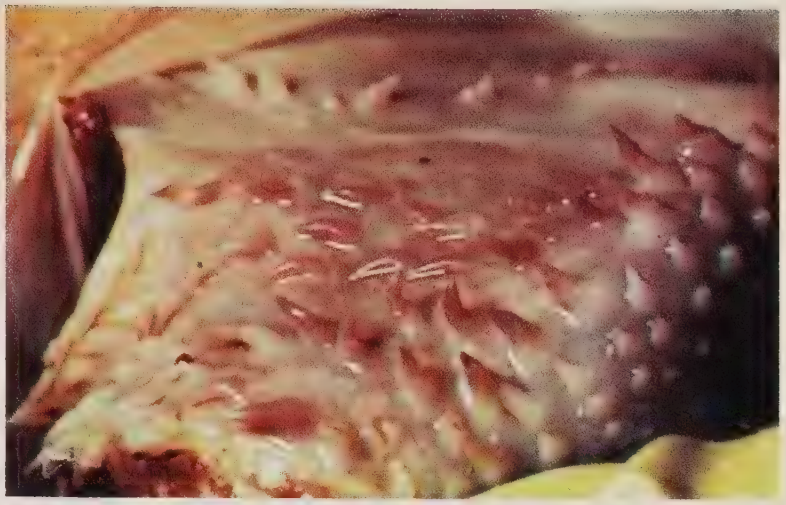
Nervous signs are rare although shivering, incoordinated movements and terminal nystagmus may be observed. Skin lesions are rare. The course of this form is usually 7-14 days.

(4) *Mild Forms:* These are syndromes caused by experimental infection of cattle using modified viruses. They are followed by recovery.

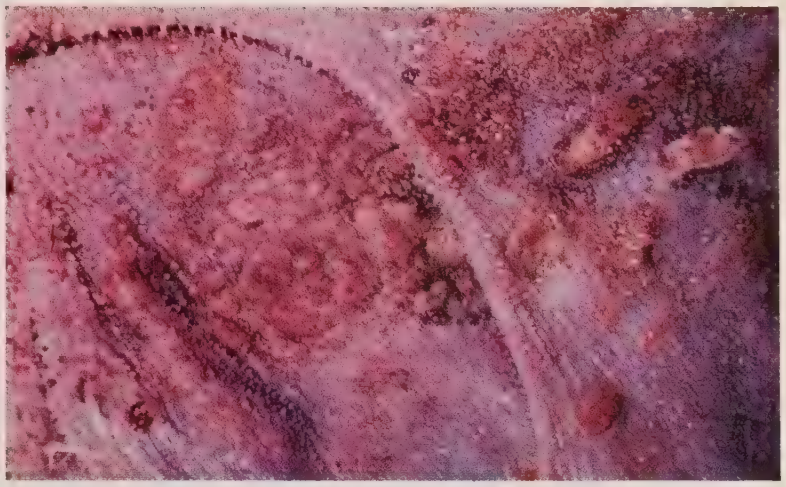
Gross Lesions: Gross lesions vary according to the form and the course of the disease. Animals that die of the peracute disease usually show no diagnostic changes.

In cases of the intestinal or head and eye form, the carcass may be normal, dehydrated or emaciated; this depends on the course of the disease. The muzzle is often heavily encrusted and if wiped, reveals an irregular raw surface.

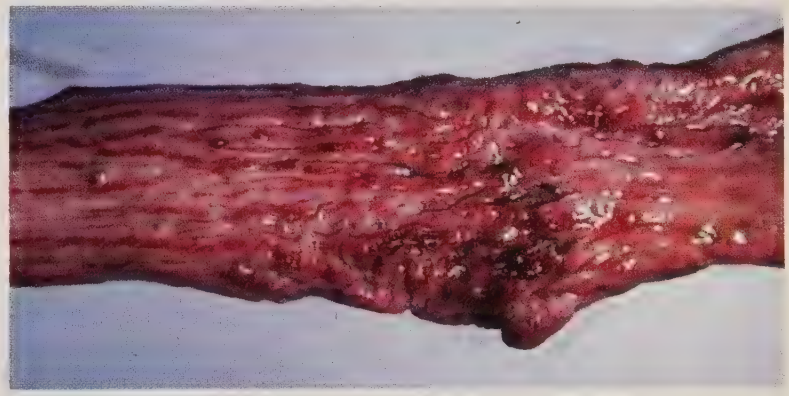
The respiratory system shows minor or severe lesions. There may be only a slight serous or copious mucopurulent discharge. The nasal mucosa shows congestion and slight to moderate serous exudate when the course is short. Later, there is a profuse purulent discharge. The mucosa is then intensely congested and edematous. Erosions may be common. Occasionally croupous pseudomembranes form; if these are removed, raw surfaces remain. Turbinates are severely inflamed and are often covered with a pseudomembranous coating. The pharyngeal and laryngeal mucosa are hyperemic, swollen and later develop multiple erosions or ulcers. These lesions are often covered in part by a cohesive greyish-yellow exudate. The tracheo-bronchial mucosa is congested and usually petechiated ulcerations occur. The lungs are normal in peracute cases but may be emphysematous in other cases. Bronchopneumonia may complicate chronic cases.



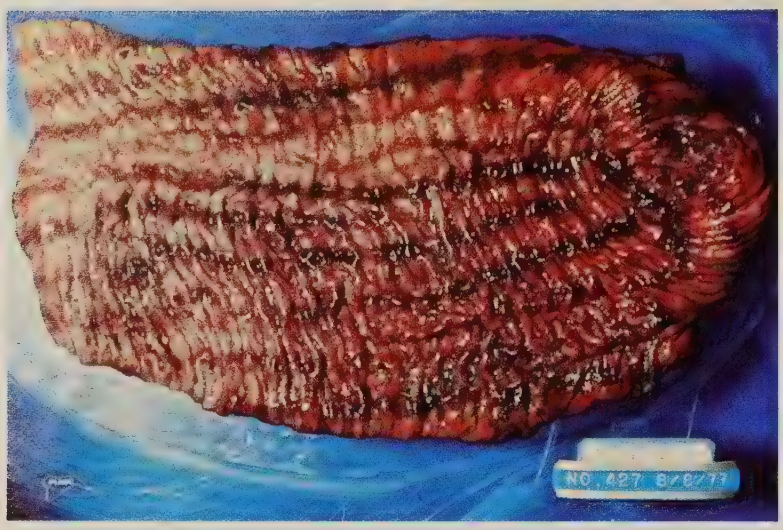
7 Necrosis and sloughing of the distal portions of some papilla (MCF).



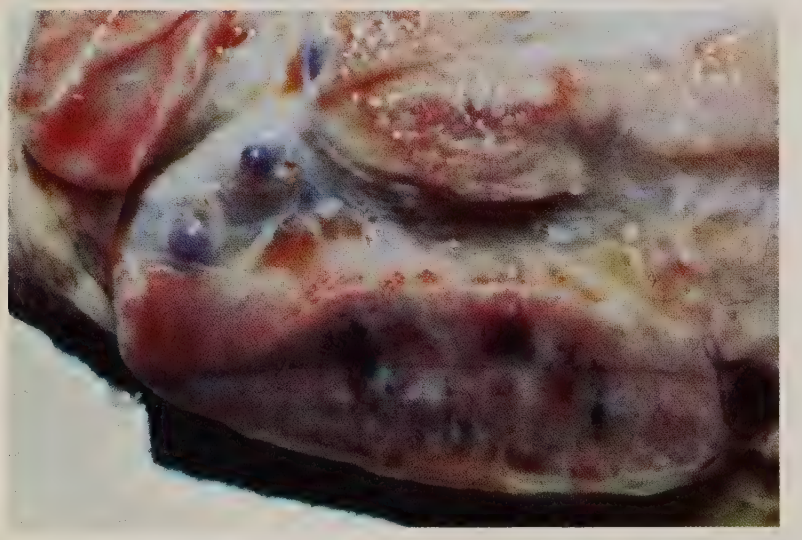
8 Lesions in the mucosa of the abomasum (MCF).



9 Erosions and hemorrhage in the small intestine (MCF).



10 "Zebra-striping" in the distal colon (MCF).



11 Edematous and enlarged prescapular lymph node (MCF).

The alimentary mucosa may show no significant lesions in the peracute disease. Hyperemia and diffuse superficial necrosis is a common feature in other forms of the disease. The erosive lesions often involve the tips of buccal papillae, gingivae, both divisions of the palate and the cheeks. The tongue is often normal. The esophagus may show congestion, erosions and pseudomembranes. The rumen, reticulum and omasum do not have lesions apart from areas of congestion. The abomasal mucosa is usually hyperemic, edematous and may have petechiae. Hemorrhagic ulcerations are also common especially in the pyloric region. The wall of the small intestine is firm and thickened by edema. The serosa may be petechiated. The first half of intestinal mucosa may show severe congestion with blood-tinged contents. These changes decrease gradually towards the large intestine. Peyer's patches are usually normal or may show superficial necrosis. The large intestines often show minimal changes, mainly lines of congestion along the longitudinal mucosal rugae. Contents of the large intestine are scant and may be dry and pasty or stained with blood.

Characteristic lesions may appear in the kidneys. They are not always seen but are typical when present. They are usually small (2-4 mm) foci of nonsuppurative interstitial nephritis. These foci form slight rounded projections from the capsular surfaces. They are whitish and represent infiltration of mononuclear cells. The urinary bladder is often normal or its mucosa may be congested. The liver is slightly enlarged and may have miliary white foci. The gall bladder is distended but normal. The spleen is often enlarged and the Malpighian corpuscles are prominent. The heart may have petechiae on the coronary groove and the endocardium may show white patches.

All lymph nodes are usually affected but the abdominal ones are less consistently involved than those of the periphery as well as those of the head and neck. Affected glands are many times the normal size, usually 2-5 times, but occasionally up to 10 times and are usually hemorrhagic. Some, including hemolymph nodes, are usually too small to recognize in the normal animal but become quite obvious when MCF strikes.

Diagnosis: A history of the disease indicating close contact between the infected animal and calving wildebeests in Africa or lambing ewes elsewhere, aids a tentative diagnosis. The long incubation period of this disease, however, often shadows the association between the natural and alien hosts of MCF. Typical clinical features help in forming a presumptive diagnosis. These include high temperature, profuse nasal discharge, severe congestion and diffuse necrosis of oral and nasal mucosa, ophthalmia, corneal opacity and gross enlargement of peripheral lymph nodes. One or more animals in a herd may be affected.

Differential Diagnosis: The clinical syndrome of MCF resembles that of other diseases especially those that cause necrosis, ulcerations and erosions of the oral mucosa of cattle. Differential diagnosis should therefore include bluetongue, bovine viral diarrhea-mucosal disease (BVD-MD), rinderpest, vesicular diseases and ingestion of caustic substances.

(1); *Bluetongue:* The clinical reactions of MCF resemble bluetongue especially in the diffuse necrosis of oral mucosa and crusting of the muzzle. Lameness

common in bluetongue is absent in MCF; ophthalmia and corneal opacity often associated with MCF are rare in bluetongue.

Virological, serological and histopathological examinations are essential for differential diagnosis of these diseases.

(2) *BVD-MD:* The classic clinical syndrome of BVD-MD occurs sporadically and is characterized by fever, leukopenia, diarrhea, lacrimation, nasal discharge and erosions of the oral mucosa.

Oral lesions in this disease, unlike those of MCF, are discrete, rounded or sharply defined depressions. Severe hyperemia and opthalmia common in MCF are not observed in BVD-MD. Diarrhea is also rare in MCF.

Final differential diagnosis requires virological, serological and histopathological tests.

(3) *Rinderpest:* Rinderpest, enzootic in Africa and parts of Asia, is exotic in this country. The clinical syndrome of rinderpest is similar to that of BVD-MD. The introduction of rinderpest virus into the highly susceptible bovine population of the USA would result in high morbidity and mortality rates, rapid transmission between animals and herds, and a disease generally more drastic than that of MCF. Mild strains of rinderpest virus could easily be misdiagnosed as the mild form of MCF.

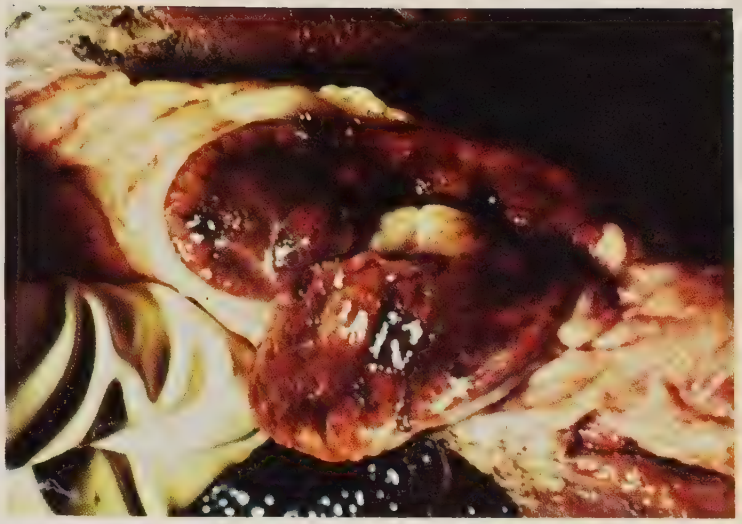
(4) Vesicular diseases, e.g., FMD or vesicular stomatitis are excluded on the grounds that these diseases elicit vesicles on the oral mucosa, teats, and coronary bands of cattle. These vesicles rupture quickly, leaving flaps of eptihelium.

Collection of Specimens for Laboratory Confirmation: Specimens required for laboratory examination in the study of MCF are:

1) Blood for virus isolation and cell count. Blood should be collected in EDTA (1 mg of EDTA per 1 ml of blood) or heparin.

2) Tissues for virus isolation: spleen, lymph nodes, adrenals and thyroids. (Blood and tissues for virus isolation should be refrigerated but not frozen and should be sent to the laboratory as soon as possible.)

3) Tissues for histopathological studies: thin slices of kidney, spleen, liver, adrenals and lymph nodes are fixed in 10% neutral buffered formalin (in physiological saline or PBS.)



12 Edematous and enlarged lymph node (MCF).

13 On cut section, lymphocytic infiltration in the kidney (MCF).

4) Paired serums are required, one collected at the onset of disease and a second during convalescence or at death.

Laboratory Confirmation: Buffy coat or cell suspensions from the tissues are inoculated onto established bovine thyroid cultures which are checked for typical CPE. Although cultures may be made from the cells of infected animals which have a high viral titer, no CPE will be observed. Cytopathic effect may also be observed by infecting bovine adrenal, kidney and testis cells as well as thyroid cells.

Animal passage may be required for final diagnosis. Viral neutralization of CPE by specific antisera may be done.

INFECTIOUS BOVINE RHINOTRACHEITIS

Definition: A respiratory disease characterized by inflammation, edema, hemorrhage, and necrosis of the mucous membranes of the respiratory passages, and pustular lesions on the genital organs of both male and female animals.

Etiology: Caused by a virus of the Herpesviridae family, which remains viable during 10 days at 37°C, but is inactivated in 21 minutes at 56°C. There are no antigenic variants although there are strains of different grades of virulence. An antigenic relationship has been detected serologically between the IBR virus and the virus of equine rhinopneumonitis.

Geographic Distribution: It has been identified in México, the U.S.A., Canadá, New Zealand, Australia, the United Kingdom, South Africa, Rhodesia, Europe, and in some South American countries.

Hosts: All ages and breeds of cattle are susceptible. The disease occurs naturally mostly in animals over 6 months of age. The disease only affects ruminants and has also been reported in mule deer, pronghorn antelope, wildebeest, and other game animals.

Transmission: The virus appears to be in greatest concentration in the respiratory tract, and nasal exudate and respiratory droplets may be considered the main source of infection. The virus may persist in a recovered animal and be discharged intermittently for up to 17 months after infection and may remain latent indefinitely following natural infection. Introduction of animals into a group often precedes an outbreak of the disease. However, it can arise simultaneously in a number of dairy farms in an area and spread from these to adjacent farms until the entire area is affected. Evidently the close confinement of feedlot cattle and large dairy herds provides favorable conditions for rapid transmission. Obstetrical operations, coitus, and licking of external genitalia of susceptible animals by IBR carriers are considered to be the most common means of transmission of the genital form of IBR.

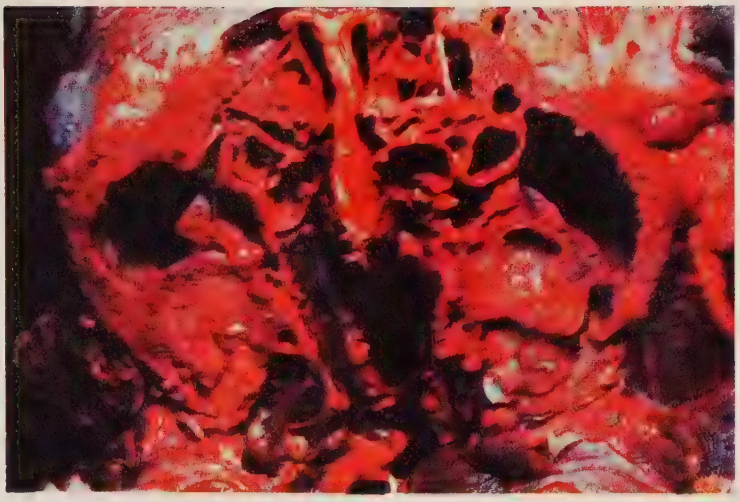
Clinical Signs: In infected feedlots the disease occurs 10-20 days after the introduction of susceptible cattle, with a sudden onset of anorexia, fever, severe hyperemia of the nasal mucosa with foci of necrosis, serous discharge from the eyes and nose, increased salivation, and a degree of hyperexcitability. A sharp drop in milk yield is seen in dairy cattle. Respiratory distress is evident, especially on exercise. Sudden

1 Breathing through the mouth and salivation in a bovine with IBR.

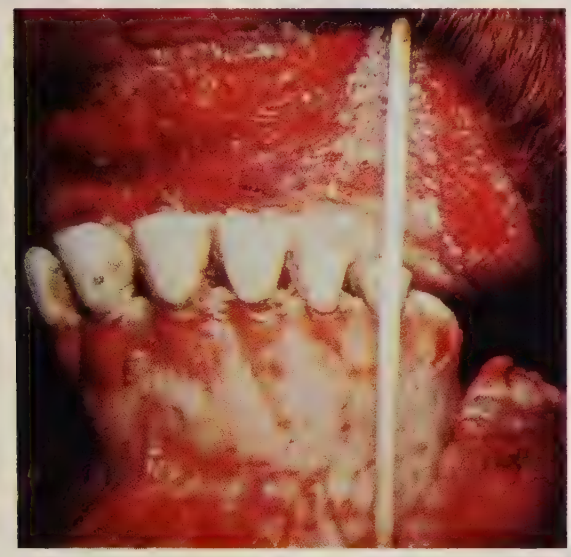
2 Ocular discharge (IBR).

3 Copious nasal discharge with hemorrhage and congestion of the muzzle (IBR).

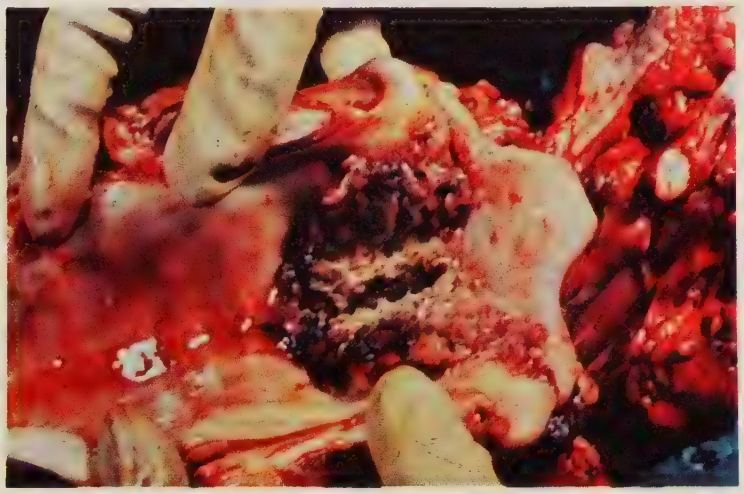
4 Close-up view of erosive lesions and diffuse hemorrhages around the muzzle-"red nose" (IBR).



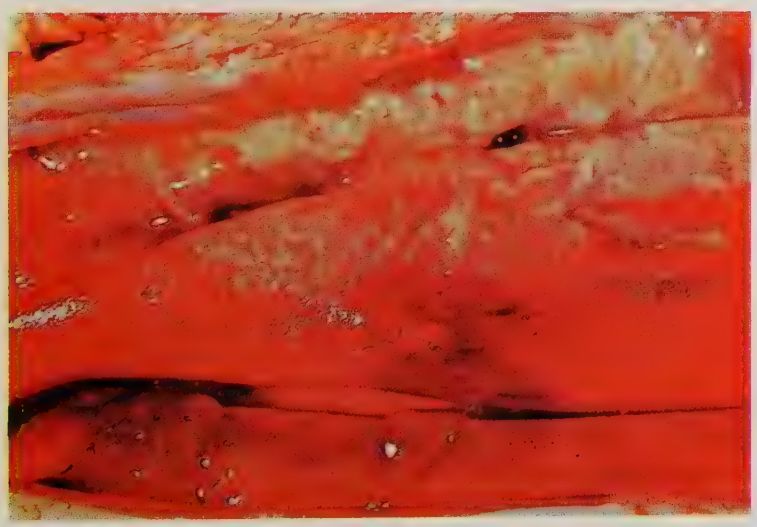
7 Cut surface of the turbinates showing diffuse hemorrhages (IBR).



5 Hemorrhages and erosions in the buccal mucosa and gums (IBR).



8 Necrotic lesions in the epiglottis (IBR).



6 Hemorrhage and exudate in the turbinates (IBR).

9 Petechial hemorrhages in the trachea (IBR).

death within 24 hours after the first signs appear can result from extensive obstructive bronchiolitis. In more prolonged cases, the nasal discharge becomes more profuse and purulent. Most fatalities are due to a secondary bronchopneumonia and in these cases a severe dyspnea, anorexia and final prostration are seen. In some outbreaks, only conjunctivitis is seen, affecting one or both eyes, with the lesions confined to the conjunctiva, without invasion of the cornea. The conjunctiva is red and swollen and there is profuse primarily serous ocular discharge. IBR may produce severe oral and gastric necrosis in newborn calves. The enteric form of the disease causes high mortality in affected calves under 3 weeks of age and chronic ulcerative gastroenteritis in feedlot cattle. The erosions found in the oral cavity with enteric IBR are also present in the rumen, abomasum, cecum and colon. Calves less than 6 months of age may develop encephalitis, which is manifested by incoordination, excitement alternating with depression, and a high mortality rate. Salivation, bellowing, convulsions and blindness have been also reported. Abortion is a common sequel and occurs some weeks after clinical illness with the respiratory form of IBR, or after vaccination of nonimmune pregnant cows with modified live-virus vaccine of bovine tissue culture origin.

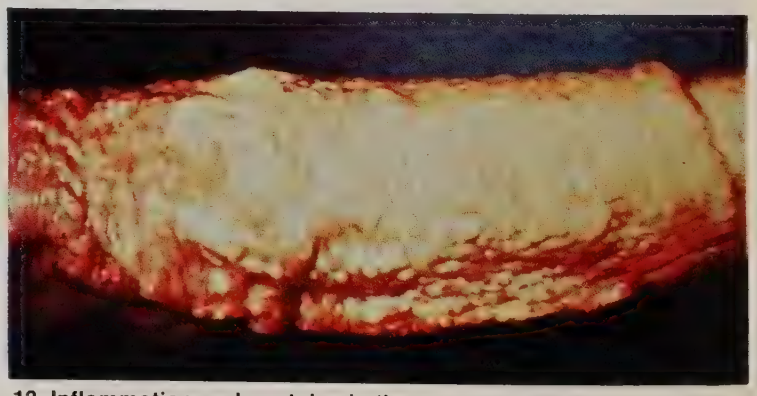
The IBR virus also produces pustular vulvovaginitis and balanoposthitis but the respiratory and genital forms of IBR are rarely found in the same herd at the same time. Evidently the genital form does not produce a viremia and does not cause abortions. In the genital form of the disease in females, the signs are elevation and twitching of the tail, frequent urination, matting of the vulva hair with blood tinged exudate, and an edematous swelling of the vulva. Reddening of the vaginal mucosa and pustule formation are initial signs. The pustules may become numerous and confluent, and a mucoid or mucopurulent exudate is seen in the vagina. In the male, inflammation of the preputial lining and glans penis, with pustule formation is seen.

Gross Lesions: In the respiratory form these are restricted to muzzle, nasal cavities, pharynx, larnyx, trachea and large bronchi. There may be pulmonary emphysema or secondary bronchopneumonia, but for the most part the lungs are normal. In the upper respiratory tract, swelling and congestion of the mucosa, petechia, and catarrhal exudate are seen. Some necrotic foci may be seen on the nasal mucosa. The aborted fetuses show focal necrotic hepatitis, and hemorrhages and autolysis in the kidney. The lesions found in the genital form of IBR have been described above.

Diagnosis: Acute rhinotracheitis with characteristic nasal lesions, bilateral conjunctivitis, fever and a gradual recovery in a few days should suggest the respiratory form of IBR. This disease should be suspected in any upper respiratory infection with sudden onset, especially if followed in 3 or 4 weeks by abortions.

10 Vaginal discharge with raised tail and continuous movement because of the intense itching in the vaginal form of IBR.

11 Congestion and pustules in the mucosa of the vagina. (IBR).



12 Inflammation and pustules in the mucosa of the penis and prepuce (IBR).

13 Diffuse hemorrhages in an aborted fetus (IBR).

Differential Diagnosis: In *pneumonic pasteurellosis* there is toxemia, lung involvement and a good response to therapy. In *bovine viral diarrhea,* and in *malignant catarrhal fever* there are erosive lesions in the oral cavity in addition to those in the nares. *Calf diphtheria* may resemble *IBR* because of the inspiratory dyspnea but the oral and laryngeal lesions and the severe toxemia are typical. In *viral pneumonia of calves* and *shipping fever* there is obvious pneumonic involvement, while in *malignant catarrhal fever* and the *mucosal diseases* lesions in the digestive tract are evident. *Allergic rhinitis* may resemble *IBR* but is characterized by sneezing and wheezing with inspiratory dyspnea, the temperature is usually normal and the nasal discharge is characteristically thickened, sometimes caseous and greenish-orange in color. In *IBR* the nasal discharge is copious, serous to mucopurulent, and discrete lesions are commonly present on the nasal septum. There should be little difficulty in making a clinical diagnosis of the conjunctival or genital forms of *IBR*.

Laboratory Confirmation: Isolation of the virus from nasal swabs using tissue culture, combined with a rise in antibody titers between acute and convalescent sera can provide confirmation of the clinical diagnosis. However, the laboratory procedures can be time-consuming, expensive and usually too late to be of much value in controlling an outbreak. Serological diagnostic techniques usually involve the virus neutralization test.

BOVINE VIRAL DIARRHEA (Mucosal Disease)

Definition: An infectious viral disease of cattle, manifested clinically by an acute erosive stomatitis, gastroenteritis and diarrhea.

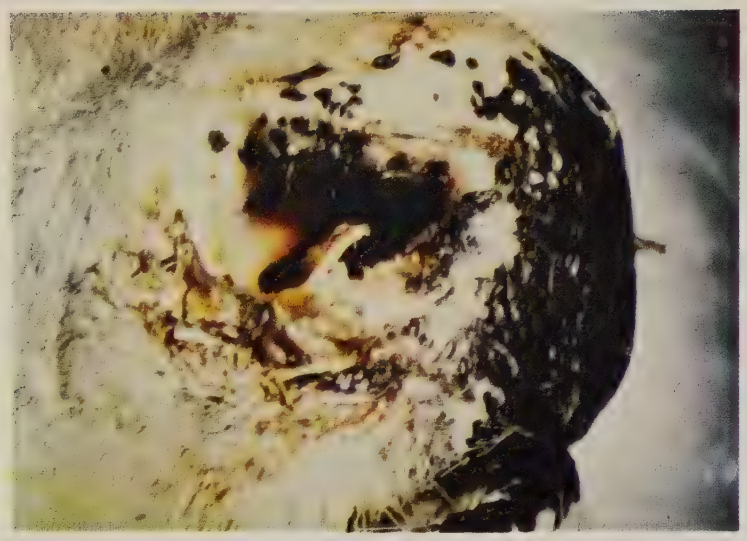
Etiology: The causal agent is one of the viruses of the Togaviridae family, of the genus Pestivirus. The virus is very sensitive to temperature, and is inactivated in a few minutes at 56°C, or in an acid pH. Antigenically three serotypes are recognized: New York, Indiana and Oregon. The latter is used in the production of a vaccine. The BVD virus is closely related antigenically to the HC virus, as shown by in vitro serological tests and in vivo cross-protection trials.

Geographic Distribution: Classic enteric BVD virus is found worldwide.

Hosts: Primarily a disease of yearlings and up to 2 and 3 year olds and occurs less frequently in older cattle. Cattle are the only species affected although a similar disease has been observed in deer and buffalo. Sheep can become infected and transmission to cattle has been shown.

Transmission: By direct contact with clinically sick or carrier animals, or by indirect contact through feedstuffs contaminated with urine, nasal or oral secretions, feces, or through contact with aborted fetuses. Transmission through aerosols or by means of insect vector is also considered possible.

Clinical Signs: The infection rate in most affected herds is high but the incidence of actual clinical disease is low, about 5%, with a case fatality rate from 90-100%. A high percentage of young cattle in an affected herd will have evidence of minor oral lesions with little or no detectable systemic illness. The incubation period is 1-3 weeks. The initial signs of the acute disease are a serous-mucoid nasal discharge, cough, polypnea, salivation, depression and fever, soon followed by a profuse, watery, foul-smelling diarrhea, which may persist for 3-4 weeks or intermittantly to 4 days. There may be erosions of the coronary band and underlying tissues of the foot may become red and swollen. Laminitis is often observed and may become chronic. Corneal opacity may occur, with an incidence as high as 10%, although usually

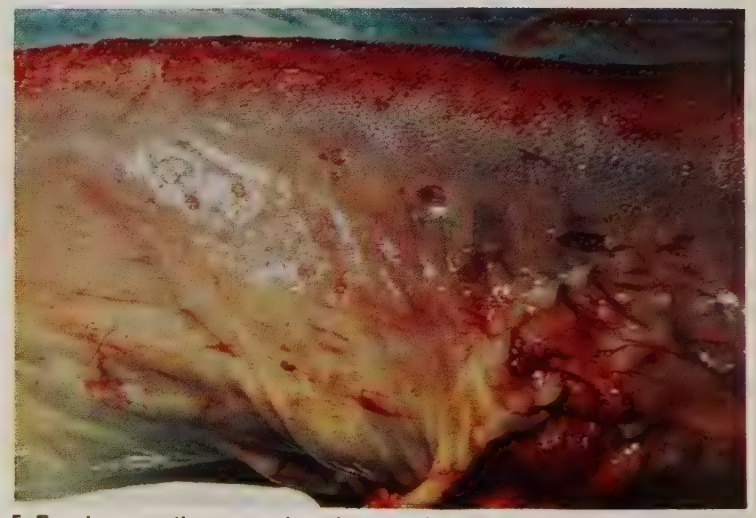


1 Necrosis and sloughing of the epithelium around the nasal orifices (BVD).

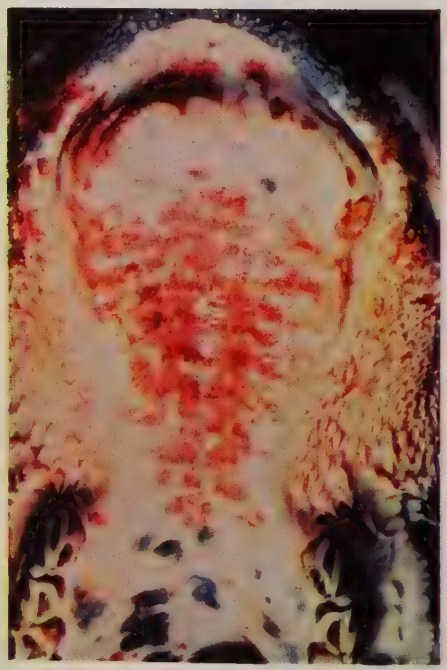
2 Erosions on the gums (BVD).

3 Erosions in the bucal mucosa (BVD).

4 Erosions on the dorsal surface of the tongue (BVD).



5 Erosions on the ventral surface and edges of the tongue (BVD).

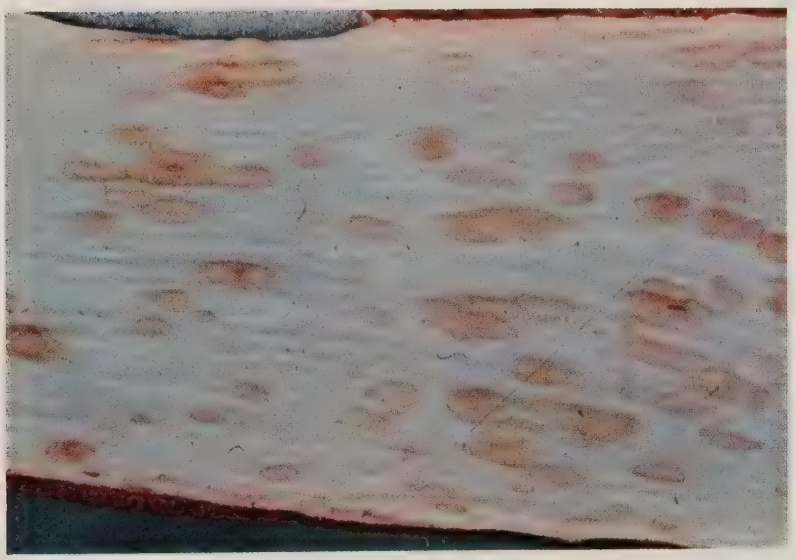


6 Hemorrhages and erosions in the mucosa of the hard and soft palate (BVD).

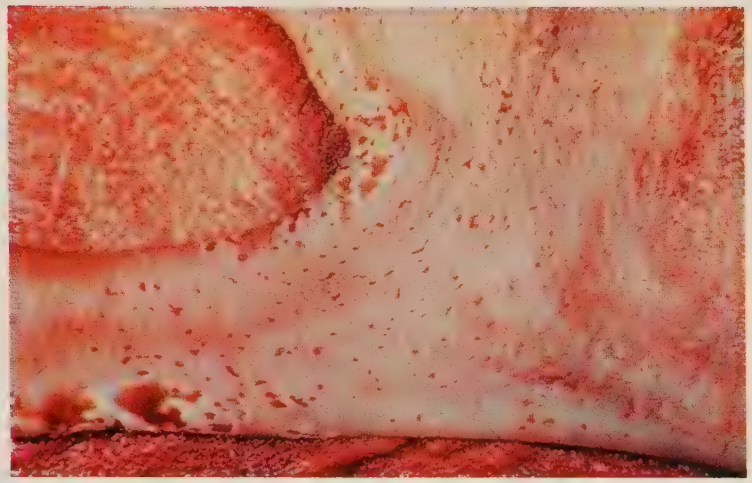
unilateral and transitory. Oral lesions are present in about 75% of the clinical cases when the animals start to scour. There is initially a diffuse reddening of the oral mucosa, then mottling of the mucosa with pin-point lesions which generally enlarge to 1-2 cm. as shallow epithelial erosions. Sites of erosions include the hard palate, soft palate, tongue, gums and commissures of the mouth. The external nares and muzzle may become hyperemic and encrusted. Abortions may be seen. The course of the disease may vary from 2-3 days up to 3 weeks and cattle with acute BVD can die in 48 hours. Frequently, affected cattle will be anorectic, exhibit oral lesions and mild diarrhea for 2-4 days, then gradually recover and come back on feed. However, if the diarrhea is profuse, the prognosis is always grave. The occasional animal that survives the acute disease is usually so badly debilitated as to be an economic liability, and will eventually die from secondary necrobacillosis or mycotic infections.

The BVD infection is also associated with congenital anomalies of the brain (cerebellar ataxia), a debilitating syndrome of young calves with arthritis (weak calf syndrome), and a chronic ulcerative disease of the alimentary tract of older cattle (Mucosal disease).

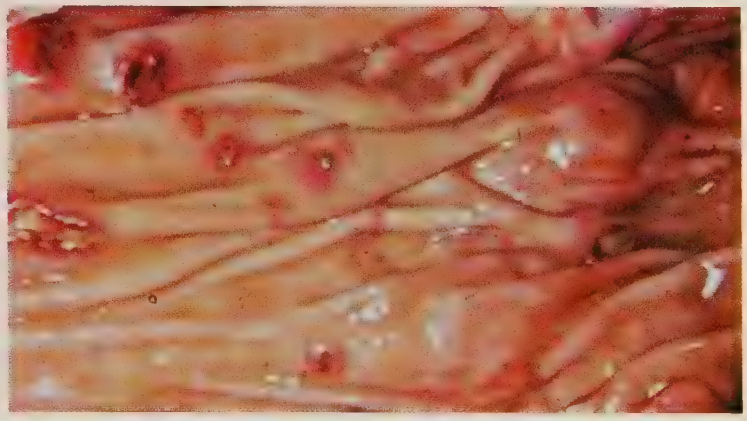
Gross Lesions: These are confined to the alimentary tract. Characteristic shallow erosions with very little inflammation around them and with a raw, red base are present on the muzzle, in the mouth, pharnyx, larynx, and posterior nares, and in the esophagus, rumen, omasum, abomasum, and cecum, and less commonly in the small intestine. There may be erythema of the mucosa and submucosal hemmorrhage in the abomasum, small intestine, and particularly the cecum and colon, where the discoloration may be marked in the mucosa folds, giving a striped appearance similar to that seen in rinderpest. The congenital defects in calves consist of cerebellar hypoplasia, cataracts, retinal degeneration, and hypoplasia and neuritis of the optic nerves.



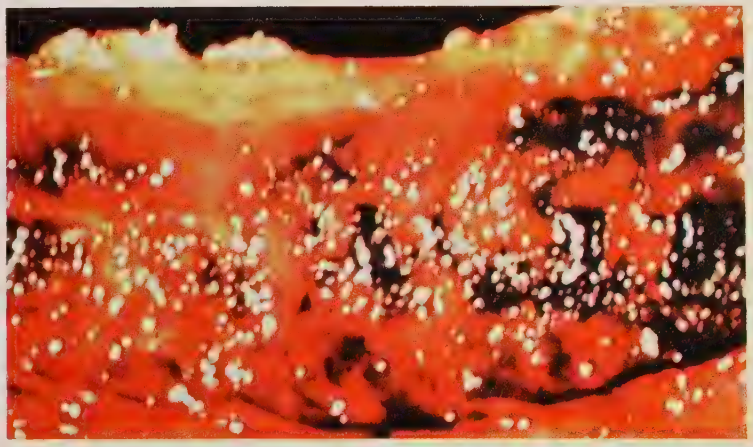
7 Erosions in the mucosa of the esophagus (BVD).



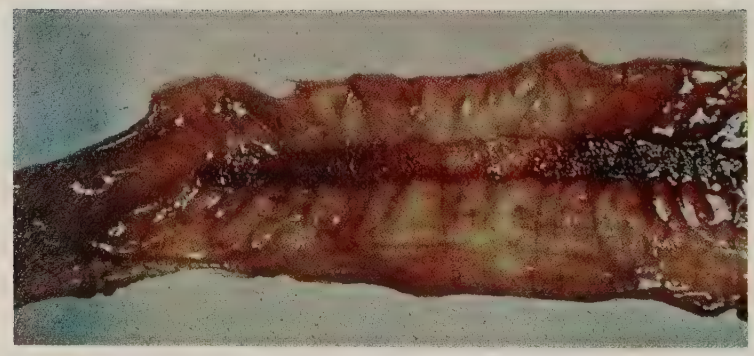
8 Congestion and erosions in the mucosa of the rumen (BVD).



9 Abomasitis (BVD).

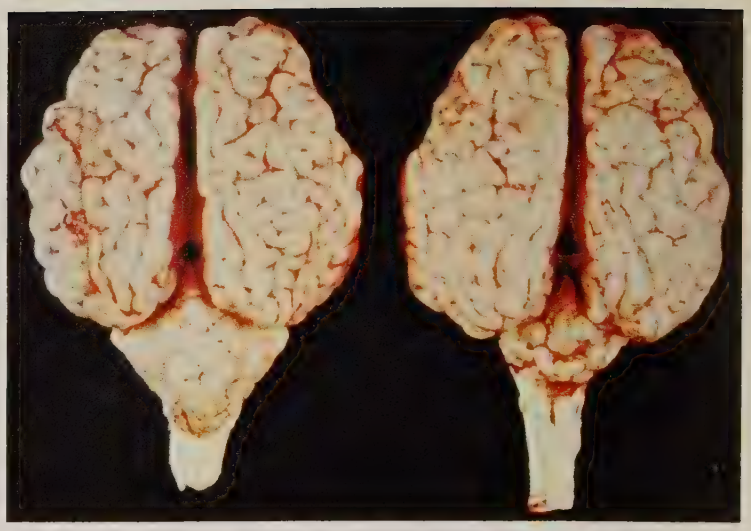


10 Hemorrhages and erosions in the mucosa of the small intestine (BVD).



11 Hemorrhages in a Peyer's patch (BVD).

12 Cerebellar ataxia in a calf infected in utero; teratogenic effect (BVD).



13 Cerebellar hypoplasia shown in BVD compared with a normal cerebellum on the left.

Diagnosis: A presumptive diagnosis of BVD-MD can be made on the basis of clinical signs, and gross and microscopic lesions; when present, oral lesions are especially suggestive of the disease. However, the differentiation of diseases causing erosive lesions of the buccal mucosa is difficult, both clinically and at necropsy, and is particularly important because rinderpest and FMD have to be considered in the differential diagnosis.

Differential Diagnosis: An erosive stomatitis and gastroenteritis are characteristic of *rinderpest, bovine viral diarrhea,* and *malignant catarrhal fever.* The stomatitis and hyperemia are particularly severe in *MCF,* and in addition, corneal opacity, which is generally bilateral and irreversible, plus a marked lymph node enlargement, especially of the prescapular nodes, and hematuria and terminal encephalitis are seen. *Rinderpest* is characterized by a high morbidity and mortality. The vesicular diseases are characterized by the presence of vesicles on the tongue and buccal mucosa, teats and coronary bands, and can be distingushed from the erosions without vesicle formation seen in *BVD-MD. Bluetongue* also produces erosive lesions in the mouth of cattle and sheep. Diseases causing diarrhea with no oral lesions include *winter diarrhea, Salmonellosis, Johne's disease* and *parasitism.*

Laboratory Confirmation: Based on isolation of the virus in cell cultures and its detection by immunoflourescent techniques. Specimens that should be submitted are feces, nasal exudate, blood and tissues collected at autopsy. Also, paired acute and convalescent sera may be examined serologically. The affected animals may have no specific neutralizing antibody because of immunosuppression or the inability to produce antibody.

CONTAGIOUS BOVINE PLEUROPNEUMONIA

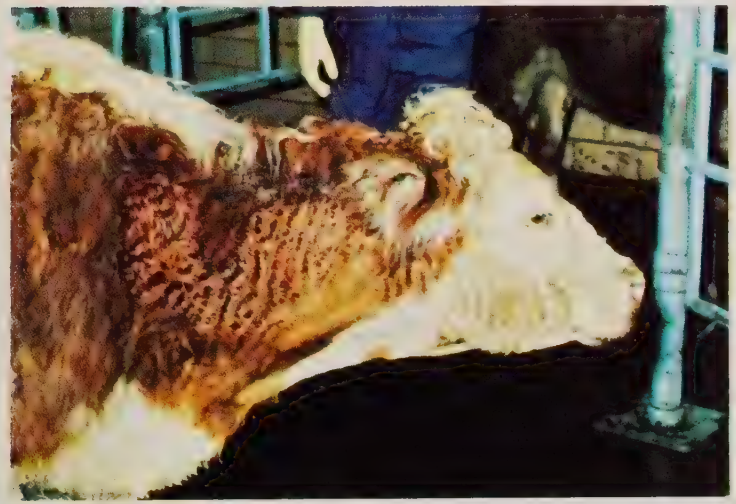
Definition: Contagious bovine pleuropneumonia (CBPP) is a specific disease of cattle causes by *Mycoplasma mycoides,* subspecies *mycoides.* It is highly infectious and occurs in acute, subacute, and chronic septicemic forms.

Etiology: *Mycoplasma mycoides mycoides* is a pleomorphic organism that is sensitive to drying heat and disinfectants. The causative organisms of contagious pleuropneumonia of goats and sheep share similar cultural and antigenic features with CBPP but are species specific.

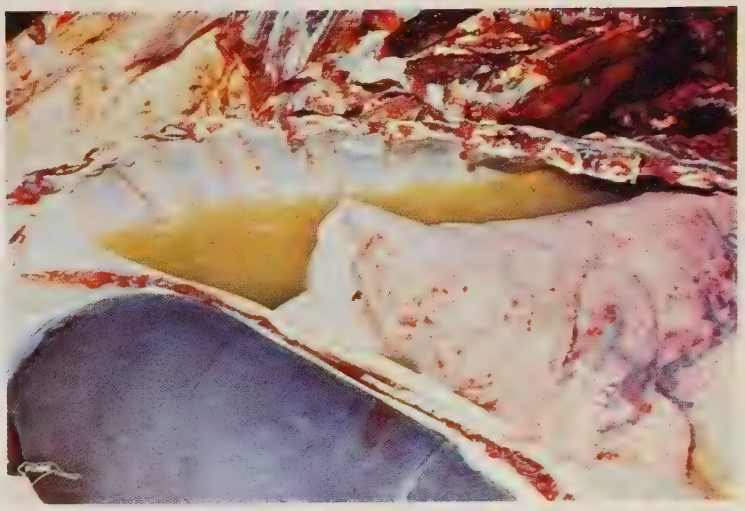
Transmission: The organism is transmitted through inhalation of bronchial secretions from infected carrier animals.

Hosts: Cattle of all ages may be infected.

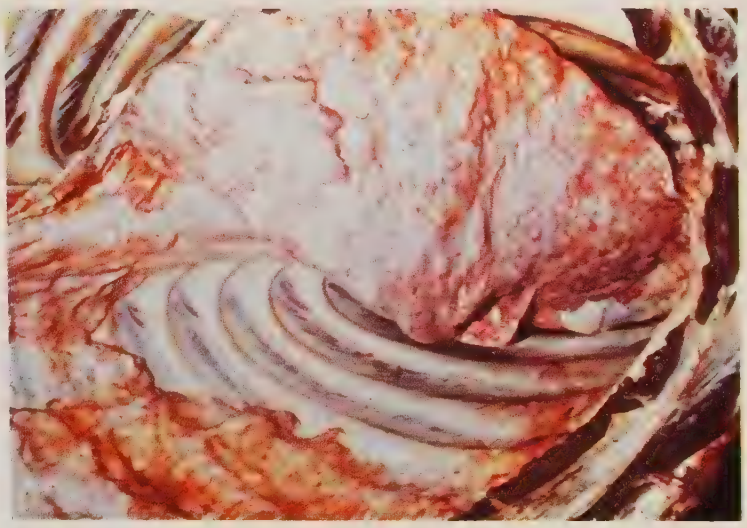
Clinical Signs: The incubation period is usually from 3 to 6 months long but it may be shorter: in highly susceptible cattle the natural disease has been known to develop in from 10 to 14 days. Initial signs are fever, cessation of rumination, and severe cough after exercise. Other signs are an arched back, chest pain, distended elbows, and extended head and neck. Grunting expiration, shallow rapid breathing with fluid sounds occur, then gurgling rales, pleuritic friction, and areas of dullness on percussion follow.



1 Bovine with contagious bovine pleuropneumonia (CBPP) stretching neck to breath easier.



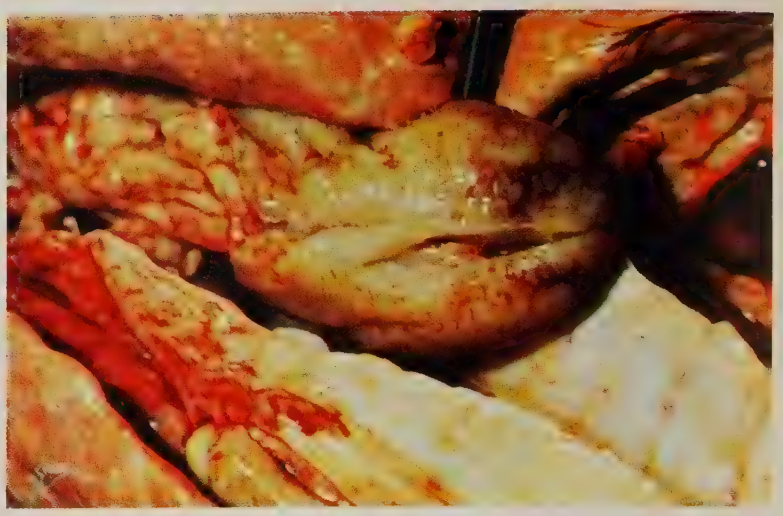
2 Straw-colored fluid in the thoracic cavity (CBPP).



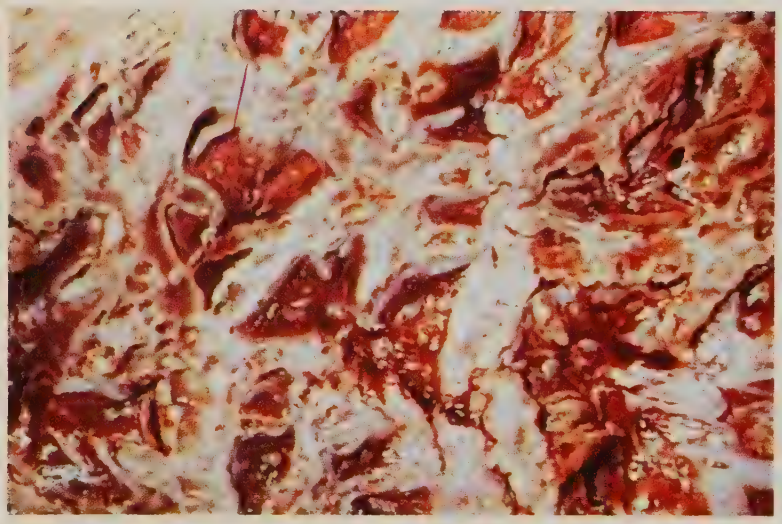
3 Pleuritis with unilateral infection of the left lung (CBPP).

4. Fibrinous mass in the thoracic cavity (CBPP).

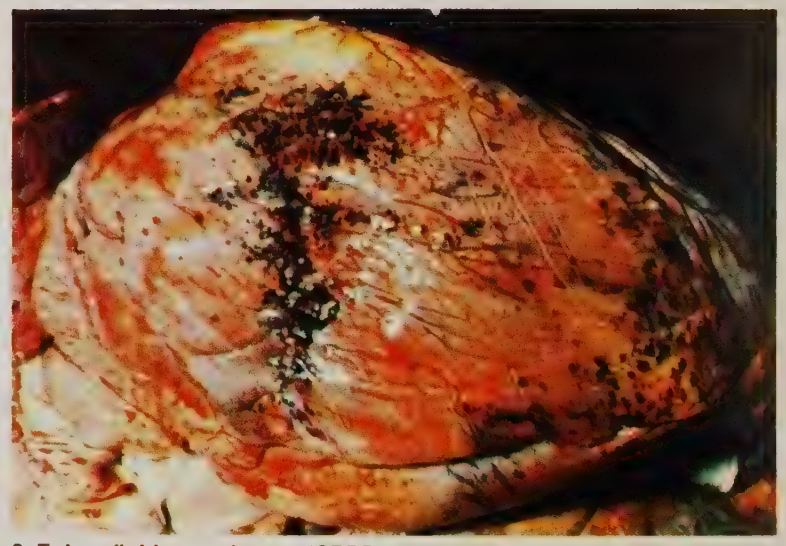
5 The interlobular septa are distended with fibrinous connective tissue demarcating lobules in various stages of hepatization; a typical lesion of CBPP.



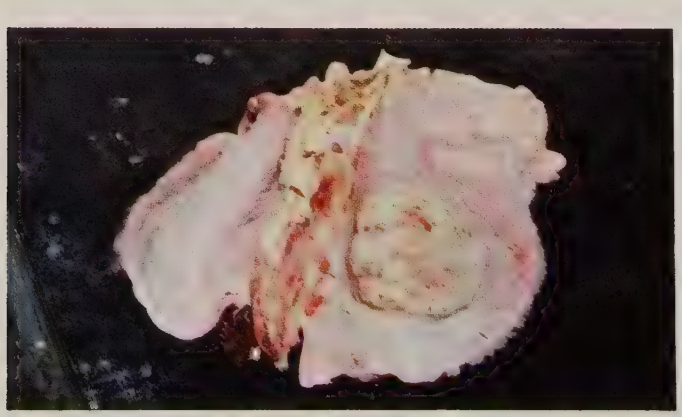
8 Enlarged mediastinal lymph node (CBPP).



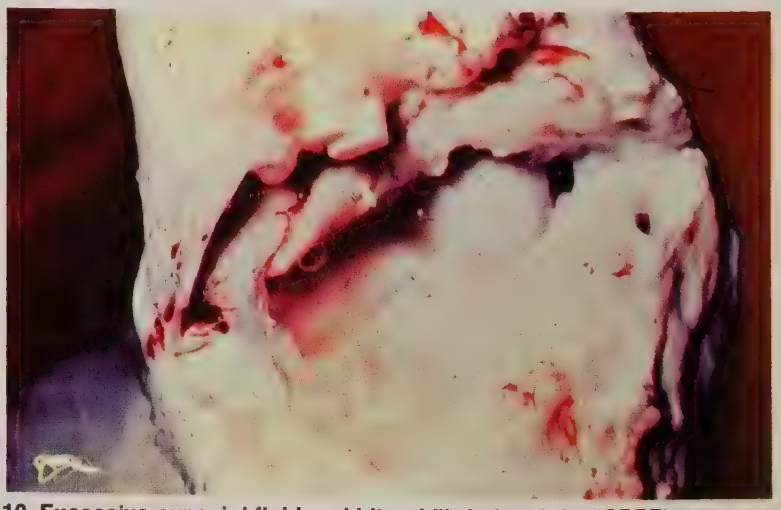
6 Close-up view showing "marbled lung disease" (CBPP).



9 Epicardial hemorrhages (CBPP).



7 Large sequestrum in the right lung (CBPP).



10 Excessive synovial fluid and bits of fibrin in a joint (CBPP).

Gross Lesions: Typical lesions found at necropsy are: thickening and inflammation of the pleura with occurrence of fibrin deposits; interlobular edema in one or both lungs. The "marbled" appearance of classical descriptions is caused by distension of the interlobular septa and is accompanied by areas of gray to red hepatization. In the chronic forms of the disease necrotic areas may be walled off by connective tissue capsules forming characteristic sequestra, which may persist for a long time.

Diagnosis: Contagious bovine pleuropneumonia is suspected when the marbled appearance of lobules and the presence of a large quantity of straw-colored fluid in the thoracic cavity are found at necropsy.

Differential Diagnosis: The lungs of animals which die of East Coast Fever may have a similar appearance to those which have CBPP. Subacute pasteurellosis sometimes may be confused with CBPP.

Collection of Specimens for Laboratory Confirmation: Samples from lung lesions, pleural fluids, lymph nodes, and lung tissue exudate are collected and frozen for isolation of the organisms. Samples from lung, spleen, brain, liver, and kidney are preserved in formalin for histopathologic examination. If possible, acute and convalescent sera are obtained.

Laboratory Diagnosis: Various serologic tests are used, including CF and agglutination. Metabolic and growth inhibition tests to identify the specific mycoplasma organism have been used successfully. The FA test is also used.

LUMPY SKIN DISEASE[1]

Definition: Lumpy skin disease (LSD) in classical (Neethling) form is an acute virus disease of cattle, characterized by the eruption of variably sized cutaneous nodules, edema of one or more limbs, and swelling of the superficial lymphatic glands.

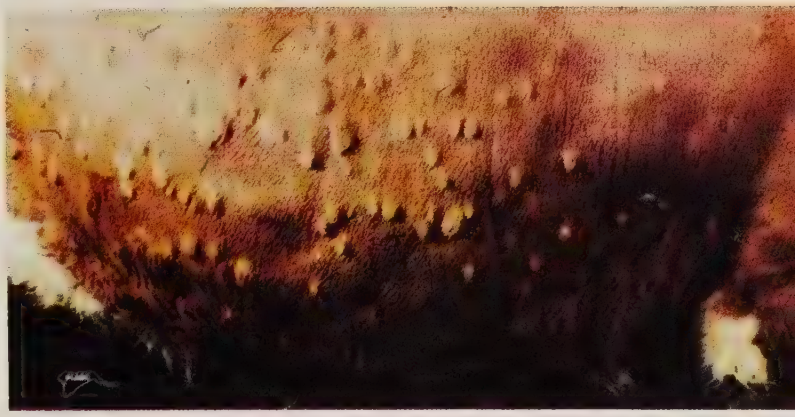
Etiology: The disease is caused by a pox virus (Neethling) that is related serologically to sheep and goat pox viruses (family: Poxiviridae).

Transmission: Insect transmission is considered more important than is contact transmission.

Hosts: Cattle and buffaloes are the natural hosts of the virus. It has been isolated and propagated in lamb testes and lamb kidney cell cultures.

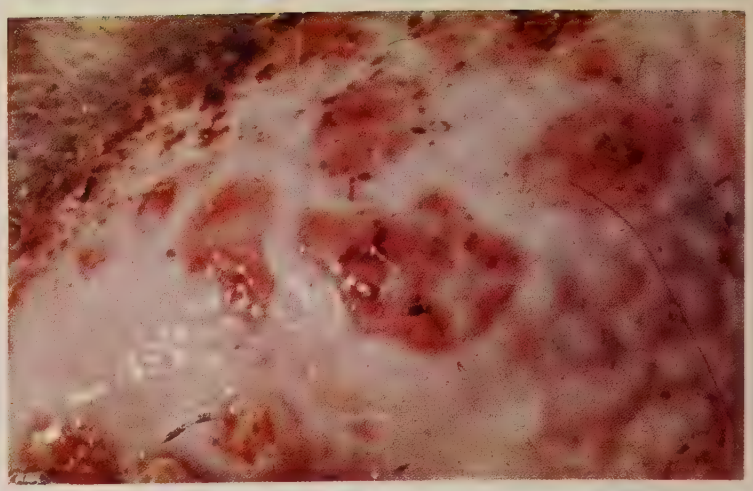
Clinical Signs: The incubation period is 4 to 14 days. There is a fluctuating fever, increased salivation, and nasal discharge. Skin eruptions occur following the peak of temperature rise. Skin nodules appear in different parts of the skin. They are easily seen on the neck, back, thighs, perineum, vulva, and around the muzzle. Lesions on the muzzle, the ventral surface of the tail, and ears are yellowish in color, covered with brownish lymph exudate and surrounded by an intensely congested zone. Mild lesions heal in a few weeks.

1 Nodules of various sizes may occur over the entire body in LSD.



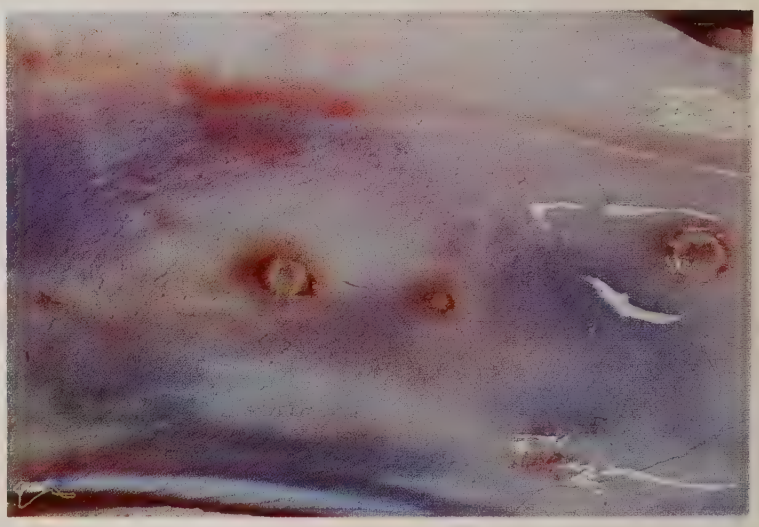
2 Nodules of various sizes may occur over the entire body in LSD.

3 Close-up view of early nodular lesions (LSD).



6 Close-up view of lesions on the nares and muzzle (LSD).

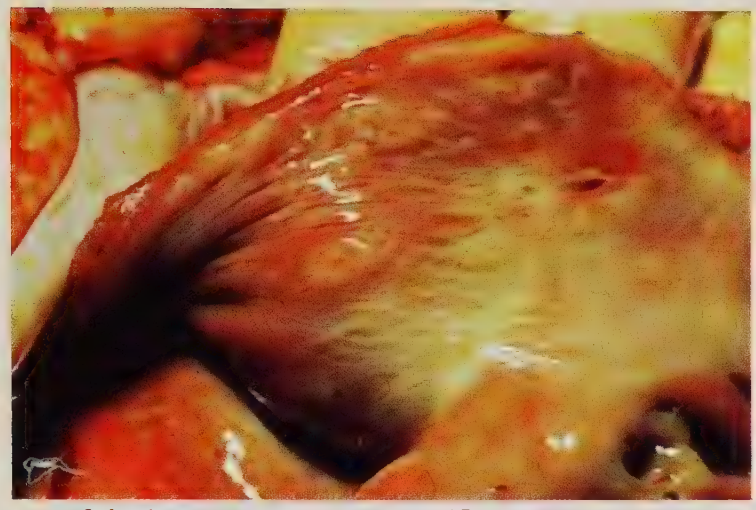
4 Nodular lesions on the tail (LSD).



7 Lesions in the nasal turbinates (LSD).

5 Lesions on the muzzle and around the nares (LSD).

8 Lesions on the mucosal surface of the trachea (LSD).



9 Lesions in the endocardium (LSD).

10 Cut surface of nodules in the parenchyma of the lung and interlobular edema (LSD).

11 "Sitfast"—a sequelae of an LSD infection.

12 Synovitis may follow LSD lesions in skin over a joint.

Gross Lesions: The skin nodules vary in size; they are thickened masses of skin tissue of a creamy gray color, sometimes containing caseous material. In mild cases the skin lesions are round, circular, and involve the superficial skin layers. Ulcerated lesions may be found on the mucous membranes of the mouth, nose, and larynx. Similar lesions may be found on the vulva.

Diagnosis and Differential Diagnosis: The nodular eruptions of the skin and mucous membranes, swelling of the limbs, and lymphatic glands are useful in making a presumptive diagnosis.

The disease must be differentiated from the Allerton type and related bovine herpes virus infections that also cause skin lesions. Other disease conditions that may be confused with LSD are allergies, screw-worm infestations, and cutaneous streptothricosis.

Collection of Specimens for Laboratory Confirmation: Fresh skin lesions should be harvested, and specimens of swollen lymph glands should also be collected and preserved on dry ice. Duplicate tissue samples are also preserved in formalin for histological examination. Both acute and convalescent sera from several animals should be obtained and frozen.

Laboratory Diagnosis: Cytopathogenicity and cytoplasmic inclusion bodies in cell cultures may be found. Inhibition of both features may be accomplished by known antiserum. Electron micrographic examination of skin tissues or cell monolayers may reveal the causative agent pox virus. Fluorescent antibody and ferritin tagging techniques are useful in identification of the virus particles in infected skin specimens and cell cultures.

BOVINE HERPES DERMOPATHIC DISEASE

Definition: Bovine herpes dermopathic (BHD) disease is made up of a group of less severe syndromes characterized by pyrexia and formation of cutaneous lesions.

Etiology: (BHD) disease is caused by herpes viruses that are similar in their biological, immunological, and physiochemical characteristics. Intranuclear inclusions, multinucleated, and giant cells develop in the skin of infected animals and cell cultures.

Transmission: The exact mode of transmission is not known; however, biting insects and milking methods are suspected of spreading the disease.

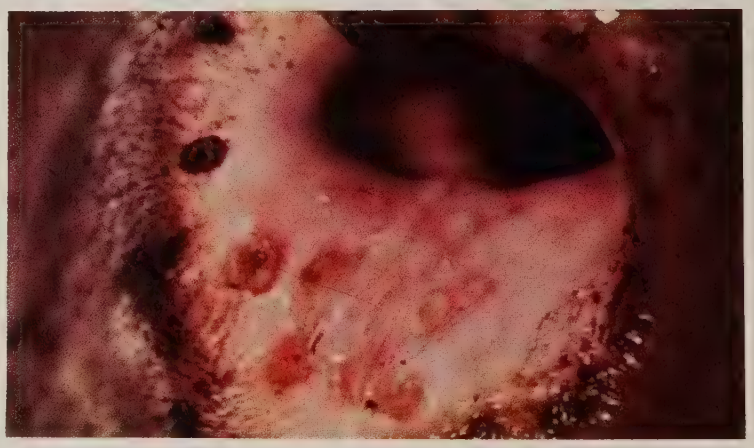
Hosts: Cattle and buffaloes of all ages are susceptible.

Clinical Signs and Gross Lesions: The incubation period is 1 to 2 weeks. A fever of several days duration precedes the formation of cutaneous nodules. The nodules are first round; later they flatten and become exudative and are covered by dry scabs. When the scabs fall off, the hairless skin is normal. Bovine mammillitis lesions are chiefly restricted to the teats and udder skin and tend to become ulcerative. A large proportion of cattle herds in enzootic areas develop neutralizing antibodies without having noticeable disease signs or lesions.

1 Dried scabs on the ear of a bovine with (BHD).

Differential Diagnosis: Signs and lesions are indistinguishable from those of lumpy skin disease and skin infections causes by *Dermatophilus congolensis,* pox, and pseudopox viruses. Lesions in the epithelium of the oral and nasal cavities cause excessive salivation,

2 Dried scabs on the skin of the neck (BHD).

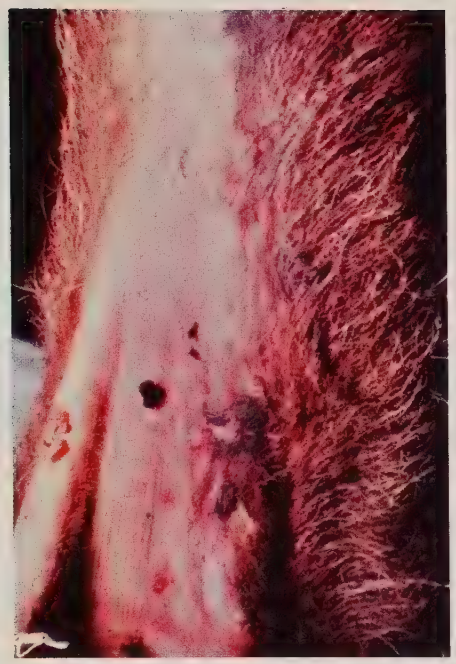


3 Lesions in an around the nostrils (BHD).

and the signs may be confused with mucosal and vesicular diseases.

Collection of Specimens for Confirmatory Diagnosis: Skin lesions may contain the virus when they are fresh and exudative. A viremia is present for approximately 4 days after appearance of skin lesions. Virus may also be obtained from vesicular fluids and from exudative teat, ear, and tail lesions. Blood samples should be taken from several affected animals in early and late disease stages to obtain paired sera. The specimens should be frozen with dry ice and sent to the laboratory

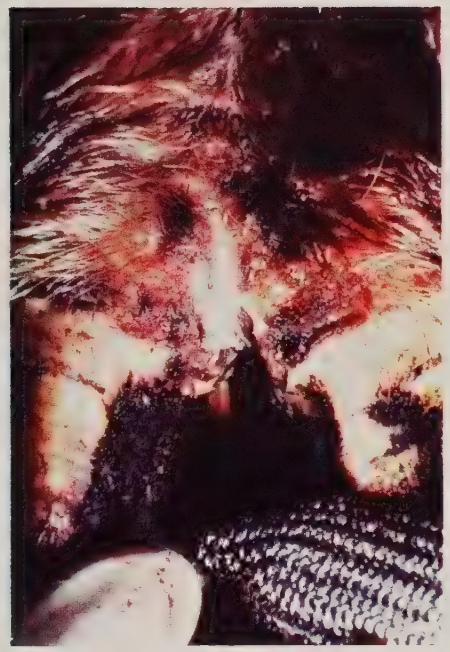
Laboratory Diagnosis: To isolate virus, fluids and skin triturates from lesions are inoculated into primary bovine kidney cell cultures. The infected tissues and



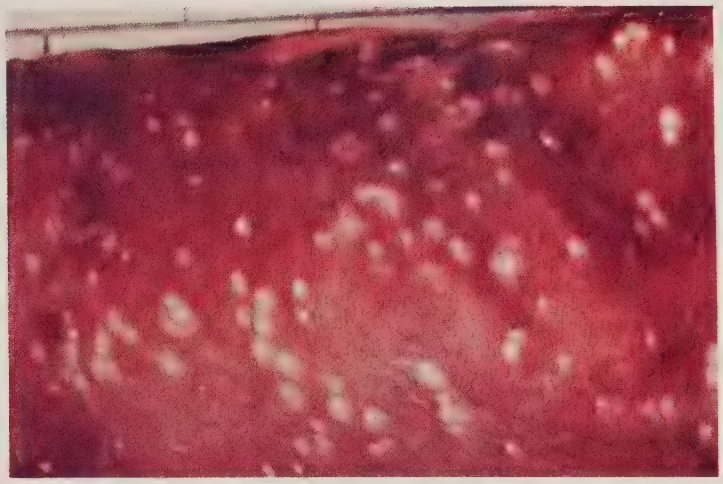
4 Lesions under the tail head (BHD).

6 Lesions on the teat with the appeance of hematomas (BHD).

7 Detachment of epithelium on the teats (BHD).



5 Dried scabs between the digits (BHD).



8 Areas of alopecia where scabs have dropped off after a BHD infection.

cultures may be examined by the electron microscope to demonstrate herpes virus morphology. Cultures stained with hemotoxylin and eosin (H and E) permit demonstration of intranuclear inclusion bodies and syncytial cytopathogenicity. The isolated virus is identified by using reference serum from convalescent animals to conduct virus neutralization and floures-cent antibody (FA) tests. Susceptible cattle, so determined by negative serologic tests for antibodies, can be inoculated intravenously to reproduce the disease and to obtain optimal tissue and blood samples.

AFRICAN HORSE SICKNESS

Definition: African horse sickness (AHS) is a highly fatal, insect-borne, febrile virus disease of Equidae, clinically dominated by an acute pulmonary edema or subacute cardiac form associated with localized areas of inflammatory edema and hemorrhage.

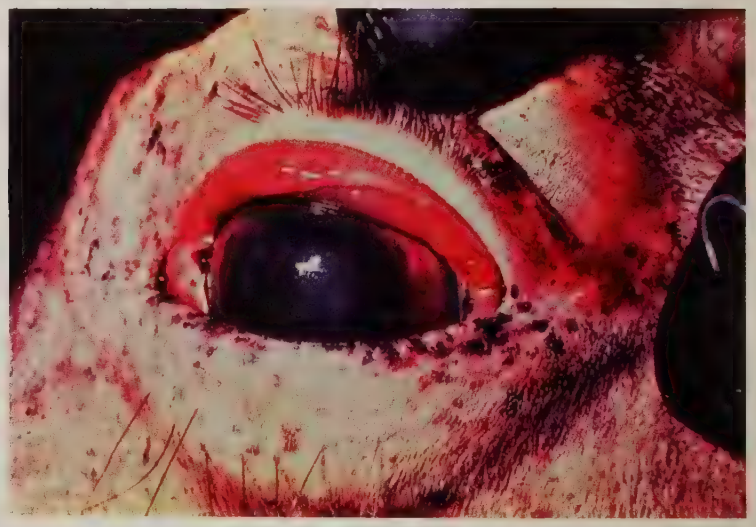
Etiology: The disease is caused by a viscerotropic virus of the family Reoviridae, genes orbivirus of which nine serologic types have been identified. Mouse-adapted strains are used for vaccines. More recently, murine virus has been propagated in cell cultures which are also useful as a source of relatively inexpensive vaccines for control of outbreaks.

Transmission: The disease is transmitted by arthropods of various *Culicoides* species. The disease may persist through seasons devoid of insects (including overwintering), as well as in the absence of Equidae. The reservoir host is not yet established.

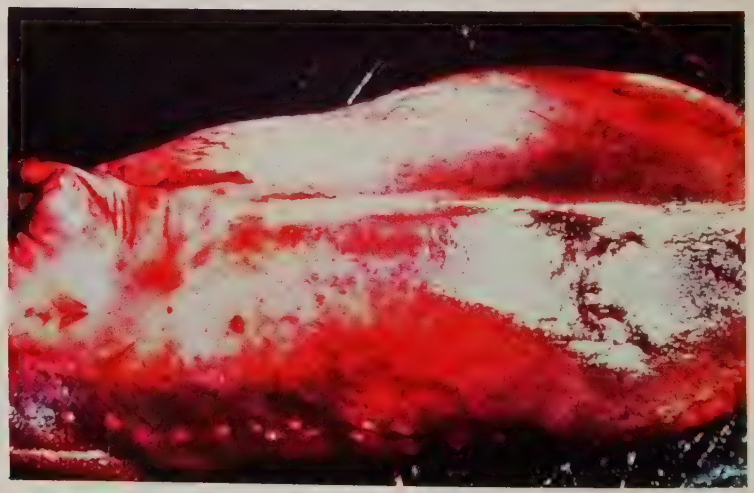
1 Frothy fluid oozing out of the nostrils of a horse in the terminal stages of AHS.

2 Ditto.

3 Edema of the supraorbital fossa (AHS).



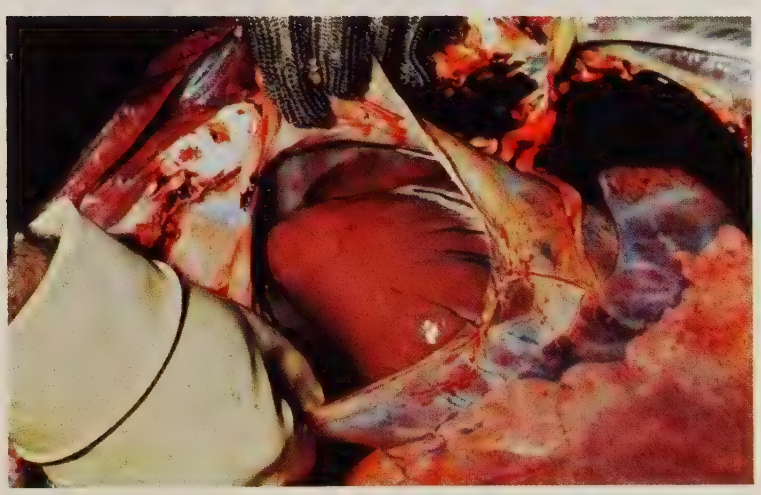
4 Congestion of the conjunctiva (AHS).



5 Ecchymotic and diffuse hemorrhages on the ventral surace of the tongue (AHS).

6 Yellow gelatinous edema in the subcutaneous tissue (AHS).

7 Yellow gelatinous edema in the intramuscular spaces (AHS).

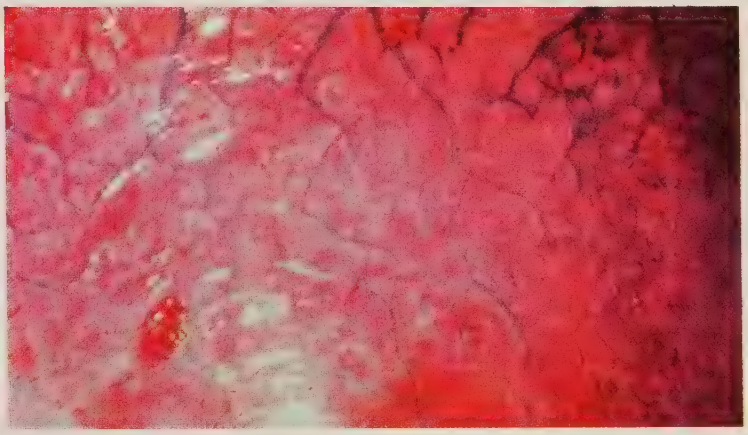


8 Pericardial fluid in the cardiac form of AHS.

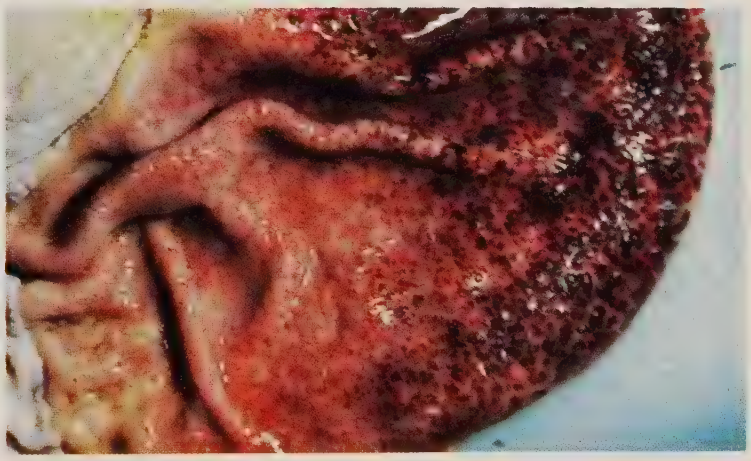
Hosts: Horses, mules, and donkeys are the natural hosts. Ferrets, mice, and dogs have been infected experimentally; dogs may become transient virus carriers after eating large quantities of infected horsemeat and blood.

Clinical Signs: When newly introduced into a susceptible equine population, the disease may appear in one of three forms: (1) a severe pulmonary disease with a 3- to 5-day incubation period; (2) a subacute or cardiac form associated with swellings of the head, neck, eyelids, cheeks, brisket, thorax, and ventral region of the abdomen (the most characteristic clinical sign is the prominent bulging of the supraorbital fossa); and (3) a subclinical form with a high temperature (104°F) for 1 to 2 days and a short period of general malaise.

Gross Lesions: The most characteristic change is gelatinous edema of subcutaneous and intramuscular tissues, especially in the region of the temple, eyes, and throat. Edema of the lungs is common, as well as endocardial ecchymoses. In some cases there are large quantities of yellowish or sanguinous fluid in the pleural cavity and pericardium. Congestion of the fundic portion of the stomach is common.



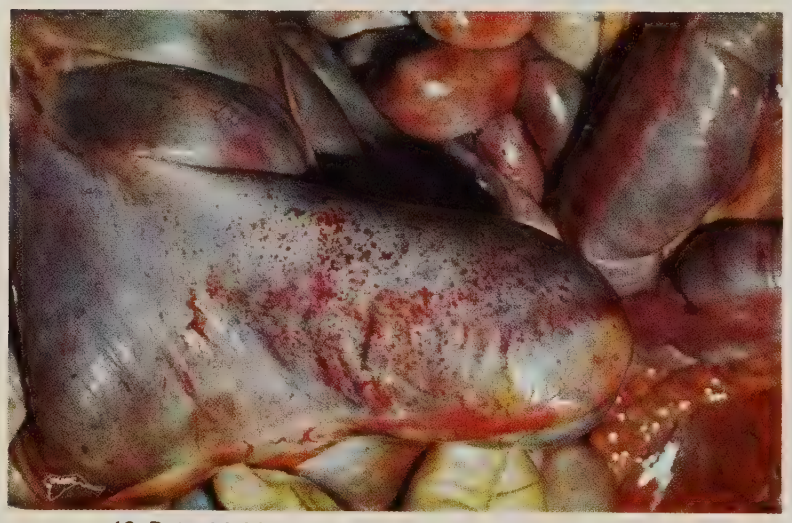
9 Interlobular edema at the margins of the lungs (AHS). Note that the lungs remain distended.



10 Congestion of the fundic portion of the stomach (AHS).

Diagnosis and Differential Diagnosis: The characteristic seasonal occurrence, history, and clinical signs may assist in reaching a field diagnosis. Signs such as edema of the supraorbital fossae, subcutaneous edema, edema of the lungs, excess of pleural and pericardial fluid, are further evidence to suspect AHS.

11 Large volumes of ascitic fluid may be found in AHS.



12 Petechial hemorrhages on the serosal surface of the cecum (AHS).

13 Petechial hemorrhages on the serosal surface of the colon (AHS).

14 Edematous mediastinal lymph node (AHS).

Diseaes that may be confused with AHS are anthrax, equine infectious anemia, and equine viral arteritis.

Collection of Specimens for Laboratory Confirmation: Blood for viral isolation should be collected in ethylenediaminetetraacetic acid (EDTA) or potassium oxylate, phenol, and glycerine (OCG) solutions (anticoagulent preservative) at or before the febrile period. Blood is also collected for serum 5 to 6 days following the peak of the temperature rise. At necropsy, a portion of the spleen is collected aseptically and may be placed in glycerol buffer. Other specimens, such as brain, heart, liver, and kidney, should be collected and refrigerated for bacterial examinations.

Laboratory Confirmation: Susceptible Equidae are inoculated intravenously with blood and spleen suspensions; these animals are observed for signs and lesions characteristic of AHS and to obtain virus and sera. The virus may be isolated by intracerebral inoculation of 2- to 6-day-old mice with diluted blood or spleen suspensions. These mice may show nervous signs in 6 to 7 days postinoculation (DPI). However, it is usually necessary to make 2 to 3 passages in mice to obtain high virus concentration in brain tissue.

The brains are harvested from mice in extremis for preparation of complement fixing (CF) antigen, which is used with reference antiserum. The test is group specific for AHS virus types. The CF antibody is at its peak in the sera of infected Equidae 5 to 6 days after the cessation of the febrile period; following this it declines rapidly. The identification of the serotypes of the virus is done by cross-neutralization tests in suckling mice or cell cultures. It is necessary to have available for these tests all nine types of virus and their homologous antisera.

Definition: Rift Valley fever (RVF) or enzootic hepatitis is an acute arthropod-borne viral disease of sheep, cattle and goats causing high mortality in young lambs, calves and kids and abortion in pregnant females.

Etiology: The virus is a member of the *Bunyaviridae* family of arboviruses although unrelated antigenically to other members.

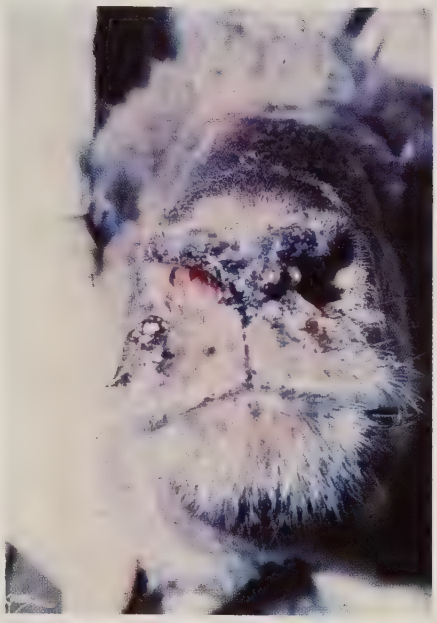
History and Geographic Distribution: Rift Valley fever was first described in Kenya in 1931, when the disease was investigated in sheep in the Rift Valley. There is strong evidence that the disease had occurred for many years previously in the Sudan and other parts of equatorial Africa. A major epizootic occurred in South Africa in 1950-51 and another in the central regions of Southern Africa in 1975. In 1977 an outbreak of RVF occurred for the first time in Egypt in the Nile Delta. In some severely affected areas, about 70% of the human population were infected. The spread of RVF was attributed almost exclusively to *Culex pipiens* mosquitoes. Except for laboratory infections, it has not been reported outside Africa.

Transmission: Daubney and Hudson, the original investigators, found that RVFV was readily transmitted to animals by blood or tissue extract inoculation. In the absence of a vector (including the needle) infected and susceptible sheep could be kept together without the disease being transmitted. Infected ewes did not infect suckling lambs.

Smithburn, Haddour and Gilbert in 1948 isolated RVFV from several species of mosquitoes in the Semliki Forest of Uganda, where there were no cattle or sheep, indicating a wildlife reservoir. In Egypt, *Culex pipiens* has been incriminated, while elsewhere, *Eremapodites* species have been involved.

Hosts: Natural infections occur in mosquitoes, sheep, goats, cattle and man. The wildlife reservoir is not yet known. A variety of laboratory hosts may be employed, including mice, ferrets, hamsters, white mice, monkeys, chicks, embryonating eggs and cell cultures employing chicken, rat, mouse, human and lamb tissues. Primary cell cultures of lamb and hamster kidney are favored.

Clinical Signs: As seen in Table I, a variety of animals are susceptible to RVF virus in varying degrees. Very young lambs, calves and kids are highly susceptible to infection with RVFV. The mortality rate is 90-100% in lambs and kids under a week old and 70% in calves. In young lambs, the first sign may be a sudden temperature rise to 40.5 to 42.2°C, followed by collapse and death within 36 hours. This acute form is less common in older sheep and goats which have a mortality rate of approximately 20-30%. Clinical signs in adult sheep and goats are not consistent but may include a rapid rise in temperature, vomiting, mucopurulent nasal discharge, unsteady gait and frequently, abortion.



1 Encrustations around the muzzle of an ovine with Rift Valley Fever (RVF).

2 A high incidence of abortion in sheep (RVF).

Pyrexia and leukopenia are customary in sheep, goats and calves, but overt signs of disease are more severe in sheep. Body temperatures of 40°C to 41.6°C for 24 to 96 hours have been recorded in calves. Clinical signs in adult cattle include high temperature, salivation, anorexia, general weakness, fetid diarrhea, a rapid decrease in lactation, and abortion. Abortion may be the only marked sign in cattle. Mortality in adult cattle is usually less than 10%. The incubation period in young lambs is 12 to 24 hours. In older animals, an incubation period up to to 3 or 4 days may occur.

Gross Lesions: The primary lesion produced by RVFV

TABLE I
HOST RANGE OF RIFT VALLEY FEVER VIRUS

4+	3+	2+	1+	—
*Lamb	Man Sheep	Goat Camel Buffalo	Cattle	Swine
*Kid	Calf (more than one week old)	Monkey (Indian & S. American)	Cat	Chickens Guinea pig Rabbit
*Calf	Rat (some species)		Dog Horse	
*Puppies			Monkey (African)	Hedgehog
*Kittens		Rat (some species)		Tortoise Frog
White mice Hamsters Fleld mice Dormouse Field mole		Grey squirrel		Canary Pigeon Parakeet

4+ = nearly 100% fatal
3+ = severe illness with some mortality
2+ = severe illness, abortion in animals, and viremia of disease
1+ = abortion, viremia, some illness to no overt signs
— = refractive to natural disease and in those species tested to laboratory infection.

* Less than one week old

Note: It is likely that with extensive laboratory testing that the very young of several species noted above would fall under the 4+ classification. Also, based upon recent data from the epizootics of the 1970's, man has been placed under the 3+ column.

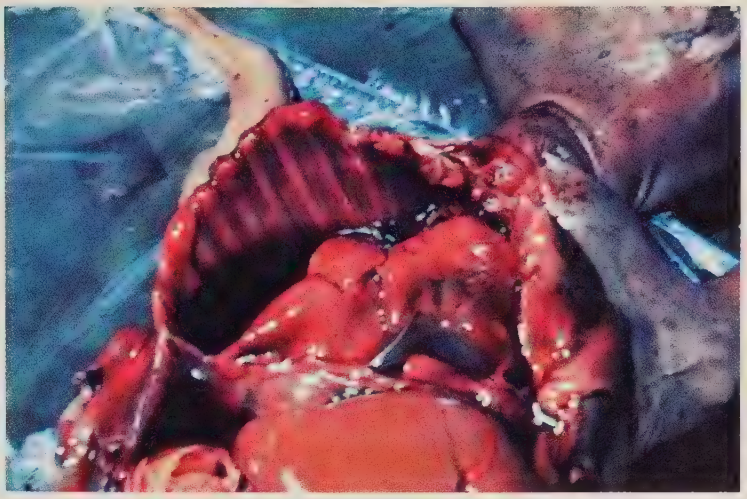
in lambs is a focal hepatic necrosis of varying degrees. At the onset of the disease, necrotic foci are associated with hemorrhages scattered beneath the liver capsule. Between 28 and 40 hours after experimental inoculation of young lambs, gray to faintly yellow foci, 0.5 to 1 millimeter in diameter, appear in the parenchyma of the liver. During the next 12 hours, these necrotic foci enlarge to approximately 2 millimeters in diamter. Just before death the liver becomes irregularly congested, focally to extensively hemorrhagic, and very soft. The primary foci may become obscured by the general hepatic discoloration. The serosal surfaces, the endocardium and the gastrointestinal mucosae remain unchanged until late in the disease, when petechia and ecchymosis may become apparent. Hepatic lesions in other species are similar to those in lambs except that necrosis tends to remain local. Hepatic lesions in adults sheep are not as severe as those in lambs but multiple necrotic areas may be present.

Diagnosis: Features indicative of an outbreak of RVF include: (1) high mortality rate in lambs, calves and kids, but a lower one in adults of those species; (2) high abortion rate among cows and ewes; (3) liver lesions at necropsy; (4) an influenza-like viral illness in man, especially in persons handling infective material; (5) suitable conditions for mosquitoes. Characteristic signs and hepatic lesions provide presumptive evidence, but laboratory methods, including virus isolation and identification, as well as various serologic techniques are necessary for a positive diagnosis.

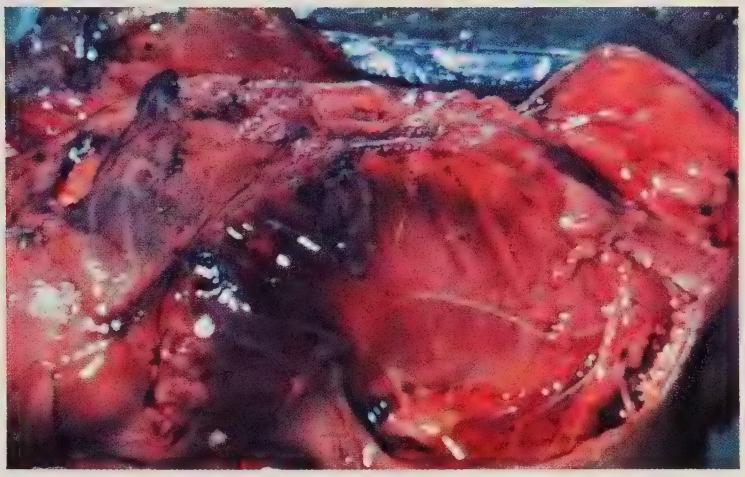
Differential Diagnosis: Rift Valley fever must be distinguished from the following diseases, among others: enterotoxemia, bluetongue, Wesselsbron and Middleburg virus infections in sheep and ephemeral fever (three day sickness) of cattle. Brucellosis, vibriosis, trichomoniasis, Nairobi sheep disease and ovine enzootic abortion should also be considered in differential diagnosis since abortion is often the only marked sign of RVF, especially in cattle. None of these diseases can be readily differentiated in the field. Laboratory methods must be used to confirm a field diagnosis or to determine the causative agent (s) of the disease.

Laboratory Confirmation: Specimens to be sent to the laboratory:

1. Whole blood at the peak of febrile response. Maintain at 4.0°C *only* if serum is not to be provided. An adequate amount for *both* virus isolation and for serum should be provided if serum is not supplied. A smaller sample of frozen whole blood may be sent for virus isolation.
2. Paired serums (or paired, unfrozen whole bloods if facilities are not available to centrifuge whole blood). One serum at the peak of pyrexia and a second at convalescence should be obtained.
3. Tissues for virus isolation: refrigerated (with ice, or at 4.0°C) or frozen liver, spleen or brain.
4. Tissue for histopathology: liver in 10% formalin.
5. Shipment: Samples other than whole blood for serum may be shipped frozen, but must be packaged to protect them from CO_2 if dry ice is used. Wet ice refrigeration at 4.0°C may be used for all specimens.



3 Macroscopically, fetuses often show hemorrhages and hemothorax (RVF).



4 Close-up view of the hemorrhages in another fetus (RVF).

Laboratory Procedures: A variety of animals may be used for RVFV isolation, including mice, hamsters, ferrets, monkeys, and baby lambs. The RVFV kills either suckling or weaned mice by intraperitoneal or intracranial injection. The yolk sac or chorio-allantoic membrane of 8 day old chicken embryos may be inoculated for viral isolation. Cell cultures may be used, but are not as sensitive as animal inoculation for primary isolation of RVFV.

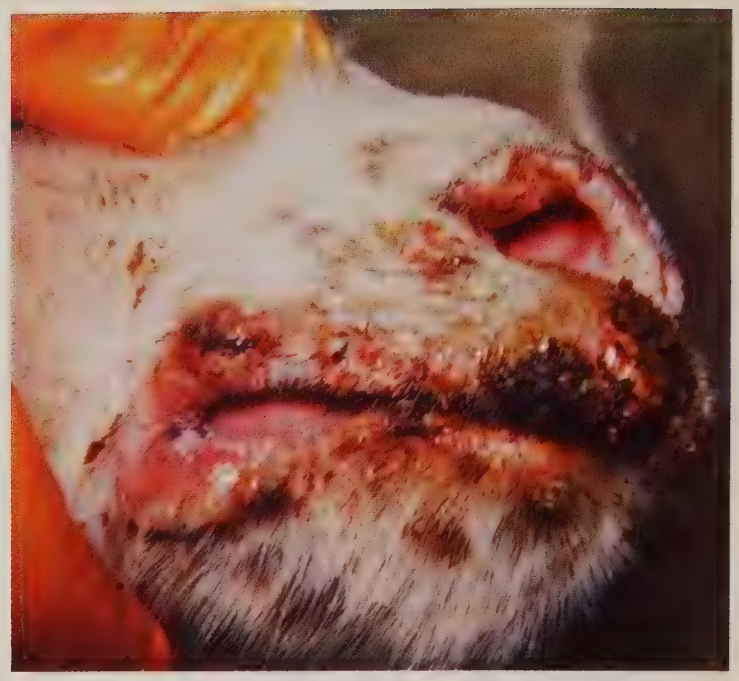
CONTAGIOUS ECTHYMA

Definition: A highly infectious viral disease of sheep and goats, charcterized by the development of pustular and scabby lesions on the muzzle and lips.

Etiology: Caused by a member of the Poxviridae family which is primarily dermatotrophic, and which has at least six distinct serotypes. The virus is immunologically distinct from vaccinia, but very similar to the causal agent of pseudo-cowpox. The CE virus is very resistant to environmental conditions, particularly dessication.

Geographic Distribution: Worldwide — occurs wherever sheep and goats are raised.

1 Proliferative lesions on the muzzle and lips of a goat with contagious ecthyma (CE).



3 Close-up view of the proliferative lesions (CE).

2 ditto.

Clinical Signs: Lesions develop initially as papules, then pustules, then as thick tenacious scabs covering a raised area of ulceration, granulation and inflammation. The first lesions develop at the oral mucocutaneous junction, usually at the oral commissures. From here they spread on to the muzzle and nostrils, and, to a lesser extent, the buccal mucosa. They may appear as discrete thick scabs, or be packed close together as a continuous plaque. Fissuring occurs in a short time and the scabs are sore to the touch. They crumble

4 Proliferative lesions around the nares and mouth of a goat (CE).

Transmission: By both direct and indirect contact. The virus resists drying and may retain viability in the scabs for months or years in unoccupied feed lots or corrals where it contaminates equipment, fences, manure, bedding and feed. Crowding facilitates direct transmission. Contamination of operating vehicles and attendants may disseminate the virus among animals of different pens or corrals. Infected suckling lambs contaminate teats and udders of dams and by this means, spread the virus among siblings.

Hosts: In sheep and goats and, rarely, in man.

5 Proliferative lesions and detachment of the epithelium on the lips (CE).

easily but are difficult to remove from the underlying granulation. Lesions may appear on the coronets and ears, around the anus and vulva or prepuce and on the nasal and buccal mucosa. In benign cases the scabs dry and fall off, and recovery is complete in about 3 weeks. Rarely, systemic invasion occurs and extends down the alimentary tract, leading to gastroenteritis, or down the trachea causing bronchopneumonia. Affected lambs may suffer a severe setback because of restricted suckling and grazing.

Gross Lesions: In severe cases, in addition to the lesions on the muzzle and lips, there may be ulcerative lesions in the nasal cavities and the upper respiratory tract, and erosions in the mucosa of the esophagus, abomasum and small intestine.

Diagnosis: In general characteristic lesions in sheep and goats are sufficient to establish the diagnosis.

Differential Diagnosis: Violent outbreaks of a very severe form may occur rarely and could be confused with *bluetongue*. *Sheep pox* may present a rather similar clinical picture but the scabs are hard crusts and there is a severe systemic reaction and a high mortality rate. In *ulcerative dermatosis* ulcers and scabs are seen on the skin of the face, feet and genital organs, but the lesions are not elevated. Among feedlot lambs or young goats the morbidity rate in *contagious ecthyma* outbreaks may be 90% or more, but mortality is low and due to secondary bacterial infection. *Bluetongue* is usually accompanied by a high mortality rate, a severe systemic reaction, lesions on the muzzle, coronets and buccal mucosa, and is more common in adults than in suckling lambs.

Laboratory Confirmation: Laboratory examination is not usually undertaken for diagnostic purposes.

SHEEP POX

Definition: Sheep Pox (SP), is a highly contagious viral disease of sheep characterized by erythematous eruptions on the skin. Early in the course of the disease, SP lesions are papular but later progress to pustular eruptions. When the lesions are generalized, they may be associated with hemorrhagic inflammation of the respiratory and gastrointestinal mucosas and high mortality.

Etiology: Sheep pox strains are immunologically identical but may vary in virulence. The virus is about 200 to 250 mu by 150 to 200 mu in size. Under natural conditions, sheep and goat pox viruses are host specific but their immunogenic relationship has been confirmed. The virus is also related to Neethling lumpy skin disease virus and contagious pustular dermatitis virus.

Geographic Distribution: Sheep pox exists in various parts of Europe, Asia and Africa. It is enzootic in Iran, India and neighboring countries. It has been reported in Egypt, the Sudan, Ethiopia and Kenya with continuing foci of the disease in Spain, Portugal and Russia.

Transmission: Transmission of SP is by contact with infected sheep, their aerosols, nasal secretions, saliva or dried scabs. The disease is transmitted by direct contact of susceptible and sick animals and indirectly by contaminated fomites and transport vehicles. The virus of SP may remain viable in wool for 2 months and on contaminated premises for as long as 6 months.

Hosts: Sheep are the natural hosts for SP virus. Other hosts have been infected experimentally. Some breeds of sheep are resistant but the Merino is highly susceptible.

Clinical Signs: The initial disease signs are fever, lacrimation, salivation and nasal discharge. Approximately 2 days later eruptions develop in the sparsely wooled areas of the skin such as the groin, scrotum, area below the tail, eyelids, lips, cheeks, nostrils, udder and vulvar labia. Sheep pox lesions begin as macules with a slight edema of the surrounding skin. Later, the lesions develop into papules which become pustules. (The formation of pustules may or may not be preceded by the development of vesicles.) As the surfaces of the pustules dry out, thin scabs are formed. The benign form of the disease is more common in adult animals; it is characterized by skin lesions, particularly under the tail, a mild systemic reaction and mortality of about 5 to 10%. Lambs commonly experience a more malignant form characterized by depression, generalized and coalescent skin lesions and frequently other lesions in the buccal, digestive and respiratory mucosae. Secondary bacterial infection may elicit a second temperature rise. Mortality in the severe form may reach 80% of the affected flock.

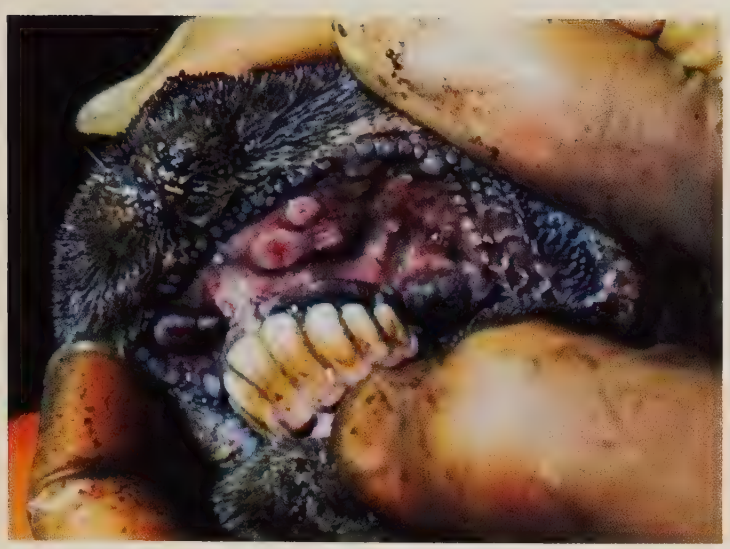
1 Sheep pox eruptions on the groin of a sheep. Note that some lesions are confluent.

2 Close-up view of papular skin lesions in a sheep (SP).

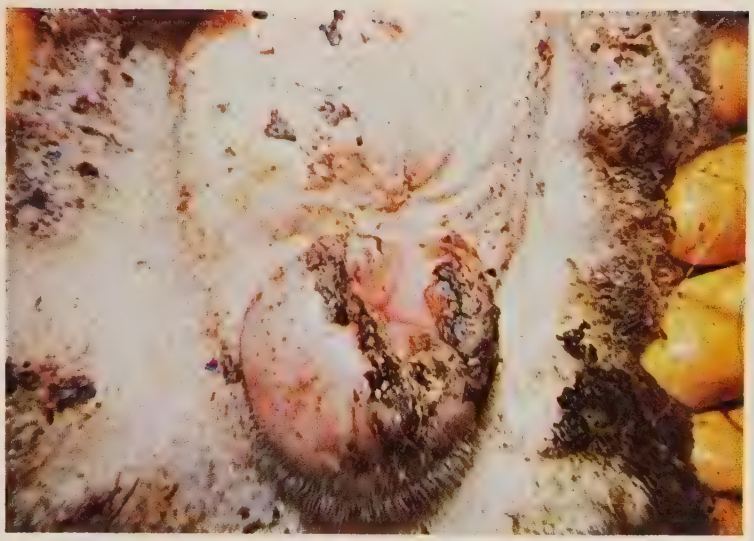
3 Lesions around the eye (SP).

4 Lesions on the muzzle and lips (SP).

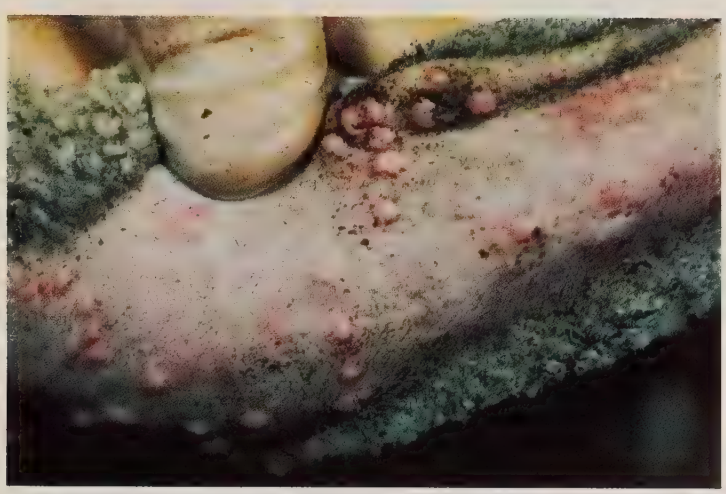
5 Lesions on the muzzle and lips (SP).



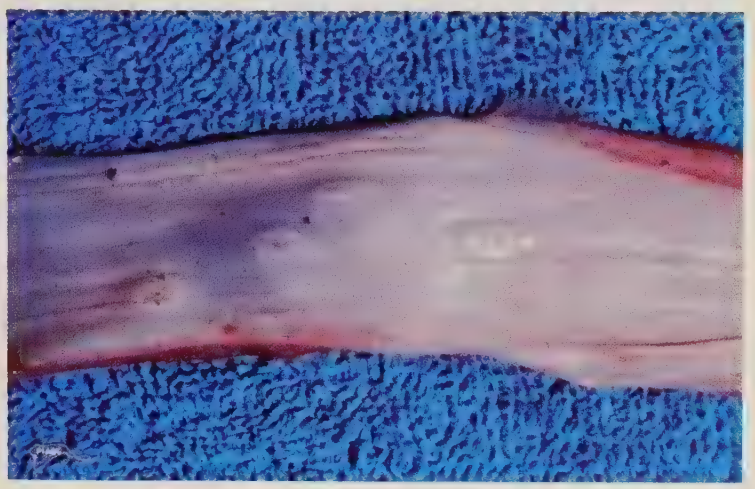
6 Lesions on the gums (SP).



8 Lesions around the vulva (SP).



7 Lesions on the ventral surface of the tail (SP).



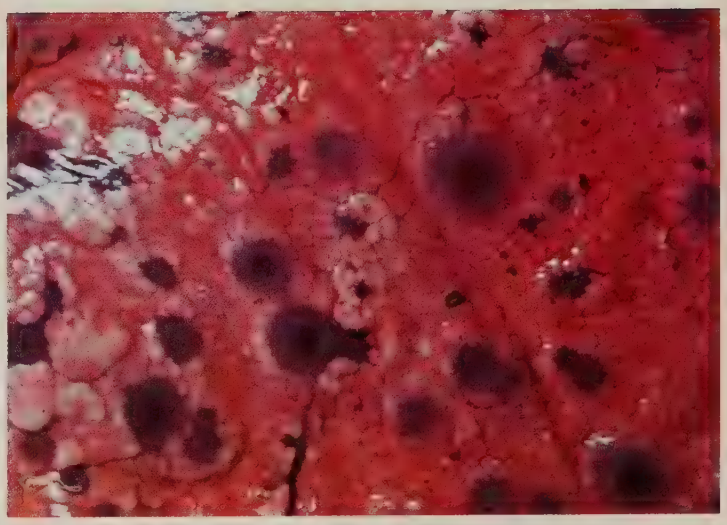
9 Lesions on the mucosa of the esophagus (SP).

Gross Lesions: The epidermal and mucosal lesions described above may be seen in the living animal or at post mortem. At post mortem the cutaneous areas surrounding the lesions are hyperemic with edema of varying degrees. All, or a combination of papules, vesicles, pustules, pocks and scabs may be found. Lesions in lambs are often coalescent. Rupture of pustules before death usually results in matting of the wool surrounding the pustule. In the malignant form, pox lesions may extend into the mucosa of the mouth, pharynx, larynx and vagina. Small grayish lymphoma-like or caseated nodules surrounded by pneumonic areas are often found in the lung.

Diagnosis: In the field, the appearance of a progressive pox or pox-like disease in a susceptible sheep flock is suggestive of SP, especially when associated with movement of animals or introduction of new stock. Clinical diagnosis of the mild form may be difficult as the lesions may be confined to small areas and be hard to detect. Laboratory assistance is necessary.

Differential Diagnosis: Formation of scab-like lesions are common to SP, eczema and scabies. Eczema is non-infectious, whereas the last is a parasitic disease. In their non-complicated form, none of them are associated with a febrile reaction. The mouth lesions and the systemic reaction in SP may be confused with those of peste des petits ruminants (PPR). Lack of papule and pustule formations on the skin and presence of necrotic ulcerative stomatitis in animals infected with PPR will assist in the differential diagnosis of the two diseases. Sheep pox may also be confused

10 Lesions in the muscle wall of the abomasum (SP).



12 Close-up view of lesions in the lungs (SP).

11 Lesions in the lungs (SP).

with contagious ecthyma (CE); however, proliferative lesions around the mouth in the case of CE as well as use of the cross-protection test may assist one in arriving at a differential diagnosis.

Collection of Specimens for Laboratory Confirmation: The following should be submitted frozen with dry ice:

1) blood from sheep taken during the febrile period, 2) lymph node and lesion material, and 3) serum obtained at the acute and convalescent stages of the disease. Portions of various skin lesions should be prepared in buffered glycerin.

Laboratory Confirmation: Direct light microscopy of stained smears from fresh lesions may reveal typical inclusion bodies. The electron microscope (EM) may be used to demonstrate the morphology of the virus and by means of ferritin tagging, the specificity of the virus. Fluorescent antibody (FA) tests are employed with various tissues. Virus may be isolated from the blood, lymph or various lesions (particularly during the viremic stage) by inoculation of cell cultures, chicken embryos or susceptible sheep. Detection of specific antibodies in the serum of recovered animals can be done by virus neutralization, complement fixation, agar gel diffusion precipitin, fluorescent antibody and other tests.

BLUETONGUE

Definition: An infectious, viral disease of ruminants transmitted by insects and characterized by congestion, erosive inflammation of the tissues of the mouth, catarrhal rhinitis, enteritis and lameness due to inflammation of the coronary bands and sensitive laminae of the feet.

Etiology: The causative agent belongs to the Reoviridae family, genus Orbivirus. The virus is highly resistant to environmental conditions and to putrefaction, being able to remain viable for long periods of time post-mortem in muscle tissue and organs. Twenty serotypes have been recognized in the world, four of these in the United States.

Geographical Distribution: Has been extensively reported in the United States, in the Middle East, Pakistán, India, throughout Africa, and is probably widespread in South America. Has not been reported in Canada, New Zealand or México, or in Europe during the last decade.

Hosts: Natural infection as determined by viral isolation in sheep, cattle, goats, white-tailed deer, mule deer, Bighorn sheep, pronghorn antelope, elk. mountain gazelle, kudu and muntjac. Serological evidence of infection in topi, black-tailed deer, bison, Barbary sheep, and camel. The disease caused by the epizootic hemorrhagic disease virus in deer is clinically identical with bluetongue in sheep.

1 Salivation in an ovine recently infected with bluetongue (BT).

2 Crusts around the nares and lips of a sheep (BT).

Transmission: Transmission is mainly by an arthropod vector, *Culicoides variipennis*. The vector reaches maximum transmission potential 10-14 days after taking blood from a viremic animal. The BT virus replicates in the arthropod, particularly in its salivary glands. BT virus is not transmitted by contact, but can be transmitted by transfer of blood from an infected animal and from an infected cow to her calf in utero. Infected bulls can shed BT virus in their semen and remain as carriers for long periods of time. Vertical transmission has not been demonstrated in sheep.

3 Intense congestion and swelling of lips and gums and sloughing of oral mucosa on the dental pad of a sleep (BT).

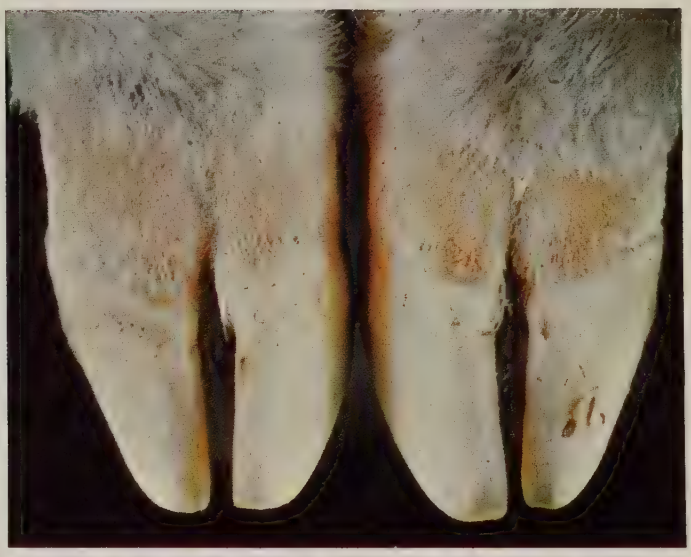
Clinical Sings: *In Sheep*-a 6-8 day incubation period is followed by depression, fever, leucopenia, salivation, oral hyperemia, congestion and swelling of mucous membranes; ulcers of lips, dental pads and occasionally tongue. Coronitis and laminitis may result in sloughing of hooves. Edema, torticollis, and vomiting are seen. Abortions and deformed lambs may be born from infected ewes. Morbidity is 80-100% in fully susceptible sheep; mortality is variable, from 0-50%.

In Cattle: Primarily an inapparent infection. Usually less than 5% of infected cattle will show any overt clinical manifestations. Mortality is very low with less than a 5% case fatality rate. Clinical signs may not be seen until 60-80 days after infection. Common signs are fever, leucopenia, myositis, salivation; hyperemia, congestion and swelling of oral mucosa and other exposed epithelial surfaces, especially the teats; ulcers on the dental pad, behind the incisor teeth and occasionally on the tip of the tongue; coronitis and laminitis, exfoliation of the epidermis and teat scabs in lactating animals; infertility and abortions, deformed and weak calves.

4 Cyanosis of the tongue and mouth (BT).

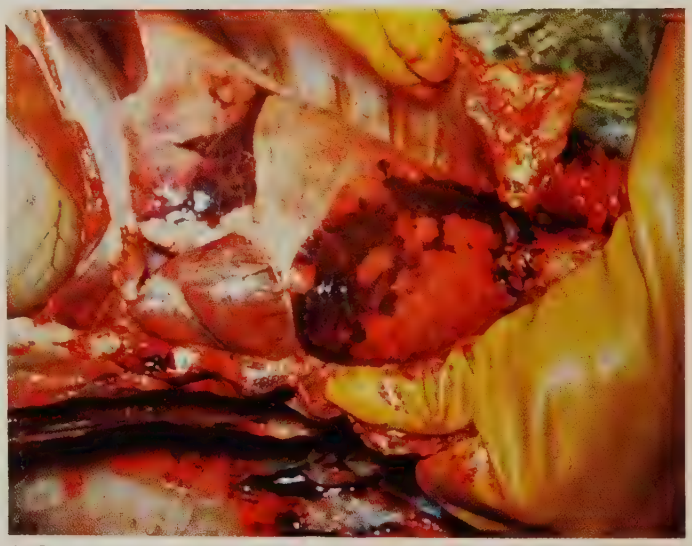
7 Close-up view of a lesion on the coronary band of a sheep (BT).

5 Severe lameness as a result of coronitis (BT).



8 Wide reddened area above coronary band of a sheep (BT).

6 Inflammation of the coronary bands of a sheep (BT).



9 Secondary Pasteurella pneumonia in a sheep (BT).

Gross Lesions: *Sheep* — Most deaths occur as a result of secondary pneumonia. Severe bilateral broncholobular pneumonia is found. A high proportion of these deaths will be associated with aspiration of vomitus. Sheep may die from acute viral infection and will show hemorrhages in the heart, edema, hemorrhage and necrosis of the skeletal muscles, enlarged, edematous, hyperemic or hemorrhagic lymph nodes and swelling, hyperemia and congestion of the spleen and liver. Congenital infection of lambs primarily causes extensive hypoplasia of the cerebellum resulting in hydrencephalus.

Cattle — Most deaths due to secondary pneumonia. In some cases cattle may die due to extensive degeneration of G.I. tract. Congenital infections of calves may cause a wide variety of abnormalities including hydrencephalus, blindness, temporary ataxia, arthrogryposis and scoliosis.

10 Muzzle of a bovine with burned appearance (BT).

Diagnosis: Seasonal incidence, cases primarily seen in late summer or early fall in temperate zones; in more subtropical areas they may be seen year-around or more often in the spring or early summer months.

Sheep — High morbidity in susceptible flocks. In enzootic areas or in previously infected flocks, only the lambs may be affected. Typical clinical pattern usually seen.

Cattle — Low morbidity-in epizootics the disease may be widespread with nearly every herd in an area having 1 or 2 clinical cases. In enzootic areas the disease is sporadic with a few cases being seen every year, but not being very widespread and involving primarily only young cattle. Mouth lesions are distinctive and are usually only confined to the anterior part of the mouth.

11 Erosion and crusts on the borders of the nares in a bovine (BT).

Differential Diagnosis: *Sheep - Contagious ecthyma:* CE lesions proliferative; BT - ulcerative. Outbreaks of CE frequently occur in fall after first frost.

Foot and mouth disease: BT does not cause vesicles. Late lesions of FMD may be difficult to differentiate from ulcers of BT.

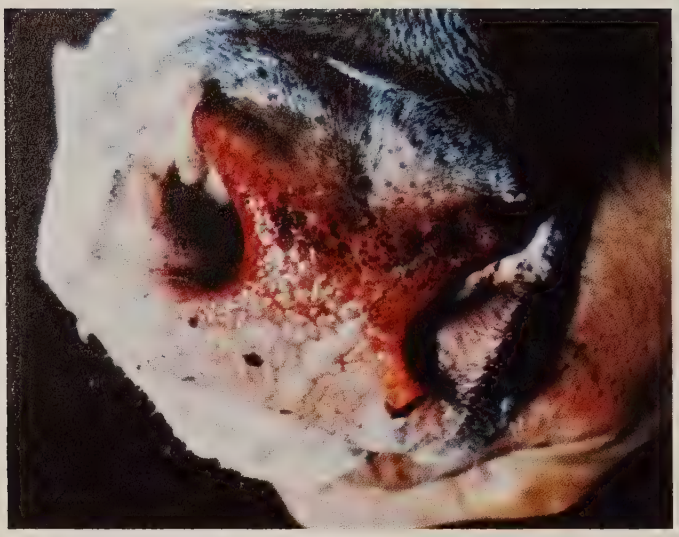
Photosensitization: May be difficult to differentiate in convalescent cases. BT may be increased in severity when animals are in sun. Mouth lesions are usually absent in photosensitization, and usually only unpigmented areas will be involved. In BT, pigmented areas will also be affected. High fevers may not be present in photosensitization.

Pneumonia: Can be secondary to BT infection. Whenever high mortality occurs due to pneumonia, BT should be considered.

Polyarthritis and foot rot or foot abscesses: Easily confused with lameness resulting from BT. BT infection and damage to the sensitive laminae of the hoof may predispose toward foot abscesses.

Sheep bots: Easily confused with the catarrhal inflamation of BT. Usually not associated with any fever or other signs of BT.

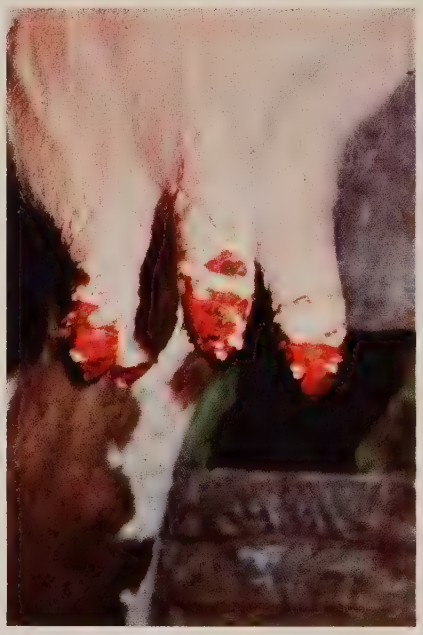
White muscle disease: May be confused with muscle necrosis often seen in BT.



12 Hemorrhage and congestion on the muzzle of a bovine (BT).

15 Intensely congested oral mucosa of a bovine (BT).

13 Severely encrusted muzzle of a bovine (BT).



16 Extensive teat lesions in a lactating cow (BT).

14 Peeling of a burnt, encrusted muzzle of a cow (BT).

17 Thickened, cracked skin in a cow, usually seen in cases with severe month lesions (BT).

Cattle-BVD-Mucosal Disease. May be difficult to differentiate. Skin lesions in BT are dry, flaky exfoliations, whereas MD usually causes a moist eczema. Cattle of all ages may have BT whereas MD is usually seen only in younger cattle. MD cases nearly all die, most BT cases recover.

IBR-Usually no respiratory involment in BT, although advanced cases may have pneumonia. IBR most prevalent after first frost.

Foot and Mouth Disease & Vesicular Stomatitis-Vesicles are not seen in BT and the spread is much slower.

Malignant Catarrhal Fever-Eye lesions normally not seen in BT, although there may be some conjunctivitis and lacrimation. MCF usually fatal. Lymph node enlargement usually not seen with BT.

Ibaraki Disease-An epizootic disease of cattle resembling BT, which has been identified in Japan.

Collection of Specimens for Laboratory Confirmation:
Samples for Virus Isolation:

1. Fresh whole blood in heparin or a sodium citrate anticoagulant.
2. Tissues freshly collected from a recently dead animal:
    a) red bone marrow
    b) spleen
    c) liver
    d) heart blood.
3. Paired sera for presumptive diagnosis of BTV infection by serological methods.

Laboratory Confirmation: *Virus isolation*-by sheep inoculation. Not routinely used anymore. Inoculation of embryonated chicken eggs intravenously. Virus isolate is established in tissue culture and identified by FA procedures.

Serological Tests: 1. Modified Direct Complement Fixation Test.
2. Micro Agar Gel Precipitation Test.
3. Plaque reduction serum neutralization test.
4. Fluorescent antibody test.

FOWL PLAGUE

Definition: Fowl plague (FP) is an acute, highly contagious, fatal viral disease of chickens and turkeys. Other birds such as waterfowl, sparrows, and pheasants are also affected.

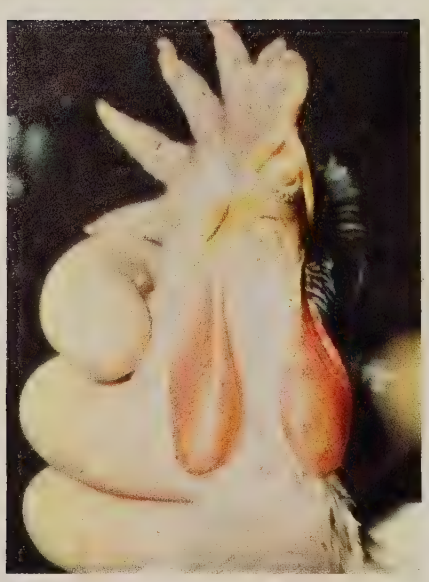
Etiology: Fowl plague is a myxovirus that can remain viable for long periods of time in infected tissues. It causes hemagglutination of the erythrocytes of chickens.

Transmission: Direct contact with aerosols from infected birds is the main method of transmission. The disease is also spread by contaminated feed and equipment.

Hosts: The chief hosts are chickens and turkeys, although other avian species are susceptible.

Clinical Signs: Depression, drooping of feathers and tail, loss of appetite, cyanosis, and swelling of the comb and wattles are common signs.

1 Edematous cyanotic comb and wattle of a chicken with Fowl Plague (FP).

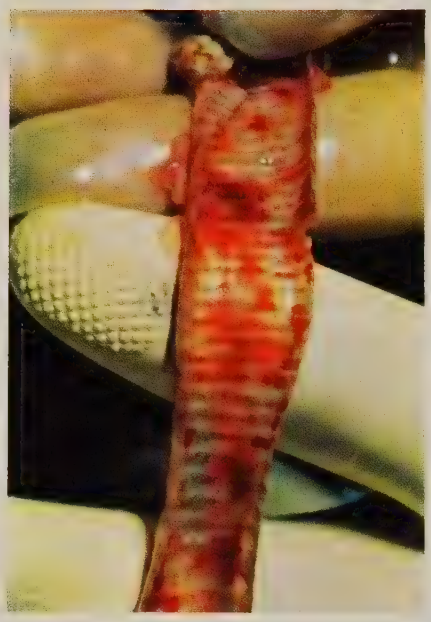


2 Edematous wattles (FP).

3 Edematous wattles dissected (FP).

4 Petechial and ecchymotic hemorrhages in the skin of the legs (FP).

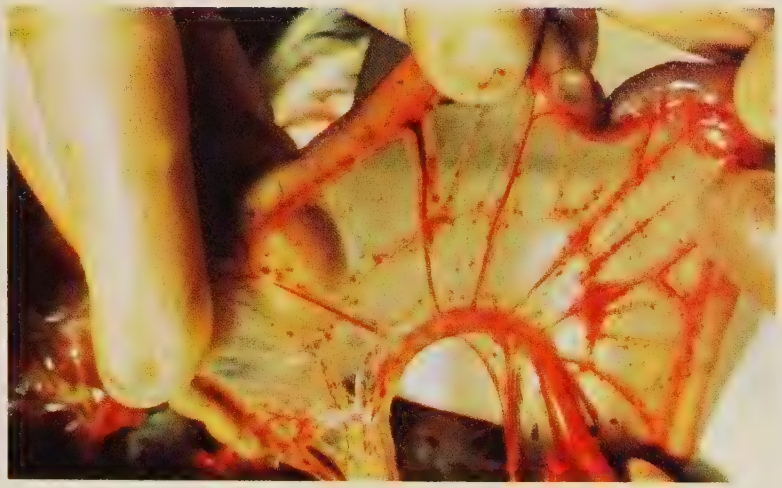
5 Bloody cloaca and dark-colored skin in a chicken that died of fowl plague.



6 Ecchymotic hemorrhages in the mucosa of the trachea (FP).

7 Hemorrhages in muscle and in the fat around the heart (FP).

8 Hemorrhages in the fat around the gizzard (FP).



11 Hemorrhage in the mesentary of the small intestine (FP).

9 Ecchymotic hemorrhages under the lining of the gizzard.

12 Hemorrhages in the small Intestine between two dark-colored ceca (FP).

10 Large hemorrhages in the fat on the serosal covering of the abdominal organs.

Gross Lesions: Hemorrhages in various parts of the body are common; these are more striking in the submucosal tissues of the proventriculus. Petechiae are found on the heart, serous intestinal surfaces, and the peritoneum. Hemorrhage in the mucous membranes lining the gizzard are also common.

Diagnosis and Differential Diagnosis: Severe mortality in susceptible chickens accompanied by the signs and lesions described previously lead to a presumptive diagnosis. The syndrome must be distinguished from virulent Newcastle disease and fowl cholera.

Collection of Specimens for Laboratory Confirmation: Specimens should be collected from several birds. Trachea, spleen, lungs, liver, and blood are the tissues of choice. These should be frozen and transmitted on dry ice.

Laboratory Diagnosis: Virus isolation is achieved by inoculation of 9-day chicken embryos. At death, the allantoic fluid is harvested and tested for hemagglutination of chicken erythrocytes. The isolated agent is then identified by hemagglutination inhibition and virus neutralization tests using specific hyperimmune serum.

NEWCASTLE DISEASE

[With particular reference to the exotic form, velogenic viscerotropic Newcastle disease, (VVND)]

Definition: A highly contagious and destructive viral disease which chiefly attacks chickens and turkeys, and is characterized by extensive hemorrhage of the digestive tract.

Etiology: Caused by a virus of the Paramyxoviridae family, which is very resistant and which will remain viable at a pH of between 2 and 12, and for 3 hours at 56°C or for 30 minutes at 60°C. The different strains of Newcastle disease virus have been classified according to their virulence: velogenic, mesogenic, and lertogenic, even though the strains are antigenically similar.

Geographic Distribution: One or more forms are endemic in India, Indochina, the Philippines, Japan, Korea, Ceylon, Kenya, Egypt, Israel, Syria, Central and South America and other countries. The velogenic strains are exotic for poultry flocks in the United States, but they are widely distributed in México.

Hosts: Domestic fowl and turkeys are the chief hosts, although other species of birds both domestic and wild, are susceptible.

Transmission: VVND is transmitted within a susceptible flock by aerosol, contact, contaminated feed, and sometimes by people such as flock attendants. Movement of apparently healthy birds in the prodromal or recovery stages can result in dissemination of the disease over long distances. The disease can also be transmitted through feeding of infected offal, feed or water. Contaminated fomites such as crates, sacks, trucks, etc. may act as mechanical carriers. Virus has been recovered from dressed poultry and this may be another factor responsible for the spread of the disease from one area to another.

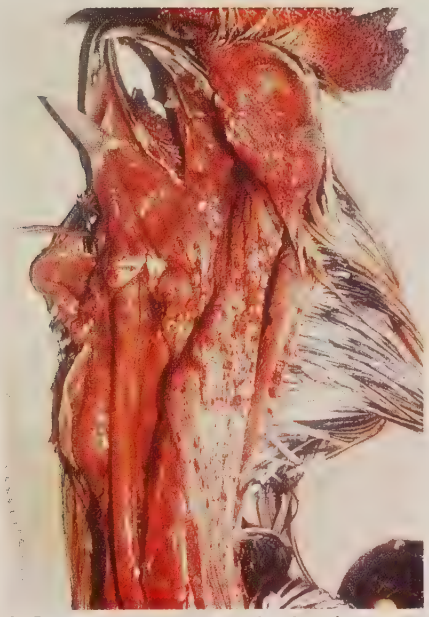
Clinical Signs: In severe outbreaks nearly the entire flock may die in 3-4 days. In the peracute form, the birds die suddenly without noticeable signs. In the acute form, the birds first appear listless, the respiratory rate increases, pyrexia appears, weakness becomes apparent and is followed by prostration and death in 5-7 days. The sick birds may display watery, greenish diarrhea which is sometimes blood stained and profuse. As a result of the fever and diarrhea, the birds appear dehydrated. Clonic spasms and torticollis may appear in birds that survive. Mortality can reach 90-100% in susceptible flocks. Some chickens may show cyanosis and edema of the comb and wattles. There may be considerable variability in the severity of the clinical signs, depending on species, age, vaccination and natural resistance of the birds, as well as the virulence of the viral strain. The incubation period is 2-6 days.

1 Drooping of the head and drooling (VVND).

2 Cyanotic comb (VVND).

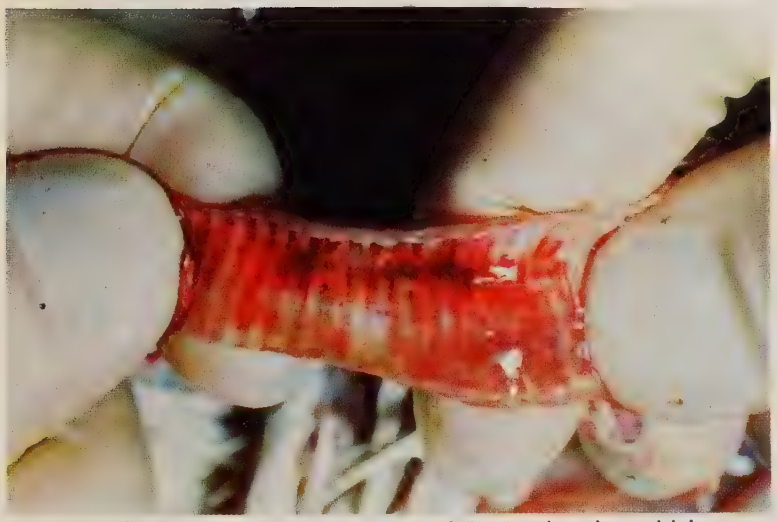
3 Conjunctivitis and edema of the eyelids (VVND).

6 Different degrees of hemorrhages in the lining of the gizzard and the proventriculus (VVND).



4 Severe hemorrhage in the deep tissues of the neck and erosions in the esophagus (VVND).

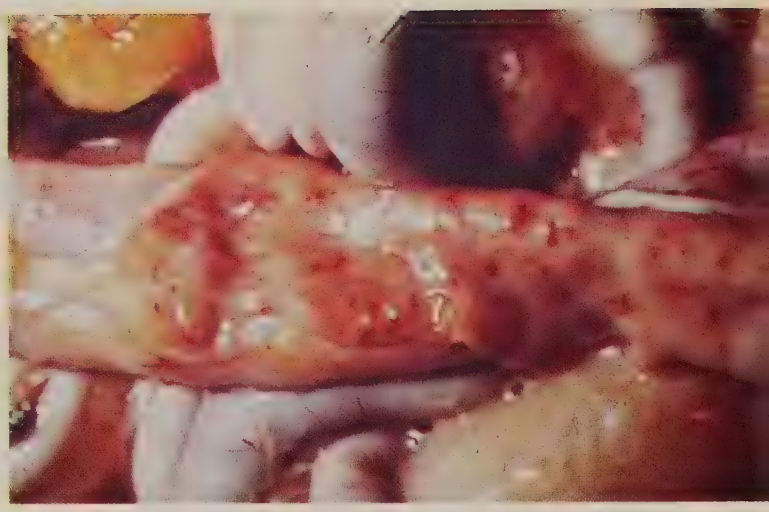
7 Ecchymotic hemorrhage in the lining of the gizzard (VVND).



5 Hemorrhage in the mucosa of the trachea in a chicken (VVND).

8 Areas of hemorrhage in the small intestine (VVND).

Gross Lesions: The mouth usually contains mucous discharges which may be tinged with blood. Dark cyanotic combs may be found on birds at death. Facial and neck edema may be severe, especially in young birds. Straw colored exudate may be excreted from the eyes and nasal openings. A diphtheritic pharyngitis may be present. Occasionally edema is present in the subcutaneous tissues of the face, at the entrance of the thorax, or at the end of the keel. The tracheal lesions are usually hemorrhagic without free blood in the lumen of the trachea. Occasionally the lining of the proventriculus is hemorrhagic as well as the serosal surface of the organ. Upon removal of the lining, the surface of the gizzard may be found hemorrhagic. Numerous small hemorrhages are frequently found in the intestines. The most constant necropsy finding in VVND is the occurence of hemorrhagic lymphoid foci in the intestines. These occur in the duodenal end and also prominently in the cecal tonsils. Lymphoid plaques or patches may be seen protruding on the surface of the intestine wall. The large intestine and cloaca may have necrotic foci. Excessive yolk-like fluid is often observed in the abdominal cavity of laying hens.



9 Hemorrhage in the cecal tonsils is the most common lesion seen in VVND.

Differential Diagnosis: The clinical signs and course of VVND closely resemble those of a number of other avian diseases, including *fowl plague, laryngotracheitis,* and the *diphtheritic form of fowl pox.* This makes laboratory confirmation of the presumptive field diagnosis mandatory.

Laboratory Confirmation: The surest method for diagnosing VVND is the isolation and identification of the causative virus. Specimens for attempting viral isolation should be selected from cases in the early or even the prodromal stages of the disease. The VVND viral strains are widely distributed in the avian body, but are present in greater concentration in the liver, spleen, blood and lungs. Tissue triturates are inoculated into 9-11 day embryonated chicken eggs, and after a variable period of incubation, depending on the virulence of the strain, the virus will be found in the chorioallantoic fluids, which are then tested for avian erythrocyte agglutinating activity. Subsequently, it is determined if the hemagglutination reaction is inhibited by known ND antiserums. Identification of virus isolates of NDV is also accomplished by serum neutralization tests in embryonated chicken eggs, and other tests such as the time necessary to kill the chicken embryo, hemagglutinating activity for red cells of different animal species and the heat-stability of hemagglutinin.

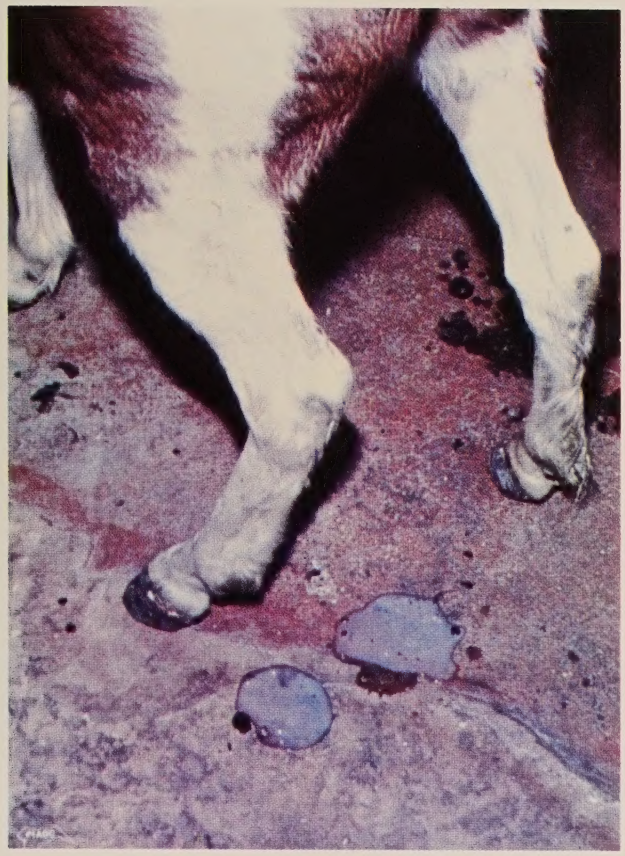
CONTAGIOUS EQUINE METRITIS

Definition: Contagious equine metritis (CEM) is a highly contagious acute venereal disease of horses that severely affects breeding and fertility.

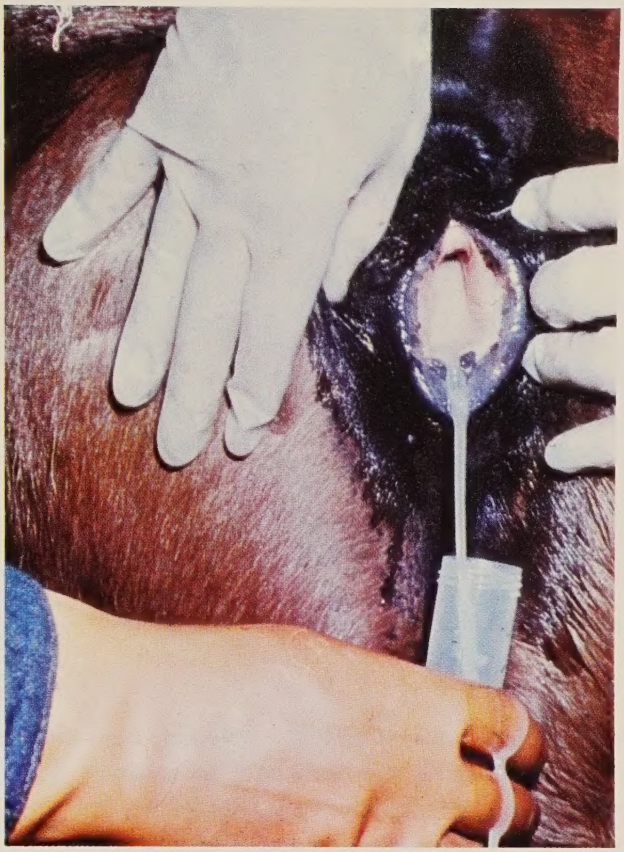
Etiology: The causative organism is not classified to this date; has characteristics of genus Moraxella and DNA base close to Hemophilus. Organism is gram-negative, non-motile coccobacillus, which is catalase & oxidase positive. It is unreactive or completely negative to standard bacteriologic differential tests. Medium for isolation: chocolate agar incubated at 37°C in atmosphere of hydrogen or air with 5 to 10% CO_2. After minimum of 48 hours incubation colonies appear as tiny pinpoints. Colonies are shiny, smooth, butyrous & slightly grey. After 72 hours, colonies appear white, raised, glistening. Resistant to streptomycin.

Geographic Distribution: France, Ireland, England, Australia, Japan, Federal Republic of Germany, Italy, Belgium, and the United States.

Transmission: Primarily venereal, but also spread by contagious means: personnel handling and examining infected mares. Rigid aseptic techniques required for



1 Mucopurulent vaginal discharge from a mare in the squatting position (CEM).



2 White, stringy, mucous exudate dripping from the vagina of a mare with CEM.

genital examination of all mares. Stud personnel who handle the genitalia of mares or studs may transmit the disease. An infected male will infect every mare covered. Fomites can transmit.

Hosts: All Equidae susceptible; to date has occurred only in throughbreds. Other species susceptibility not known.

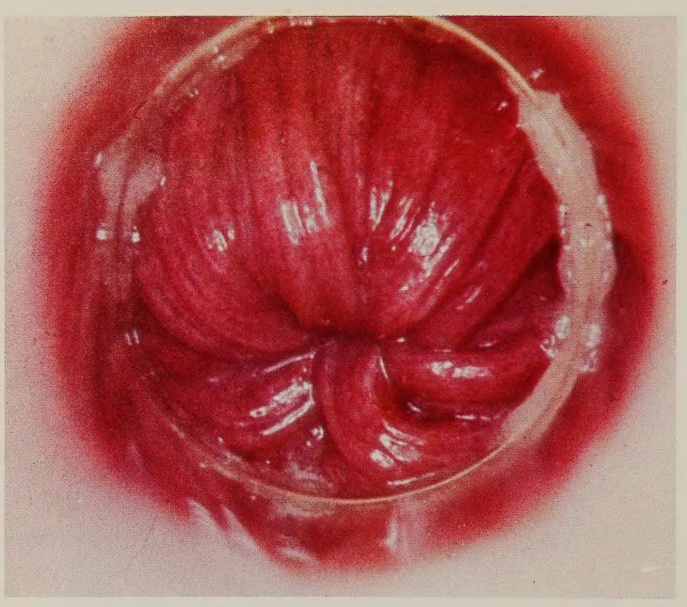
Clinical Signs: First indication to clinician is a history of each mare covered by a particular male coming back into premature heat; each such mare will appear "dirty" on vaginal examination. Routine culture reveal nothing except Proteus spp. Infection is characferized by endometritis, inflammation of cervix and vagina. Copious mucopurulent discharge sometimes seen coming from the vulva, smearing buttocks, matting tail. Severity varies from complete sloughing of endometrium to mares which appear normal, except for premature heat; may or may not have small volume of greyish fluid on floor of vagina. Stallions are completely normal. Asymtomatic carriers known.

Gross Lesions: Few post mortem examinations have been made to date. Gross lesions varied from slightly enlarged uterus with small amount of greyish fluid to grossly enlarged and turgid uterus with an opening which appeared to be a pyo-mucometra. This uterus was full of greyish mucopurulent fluid & the endometrium had a very "glossy" and thickened appearance.

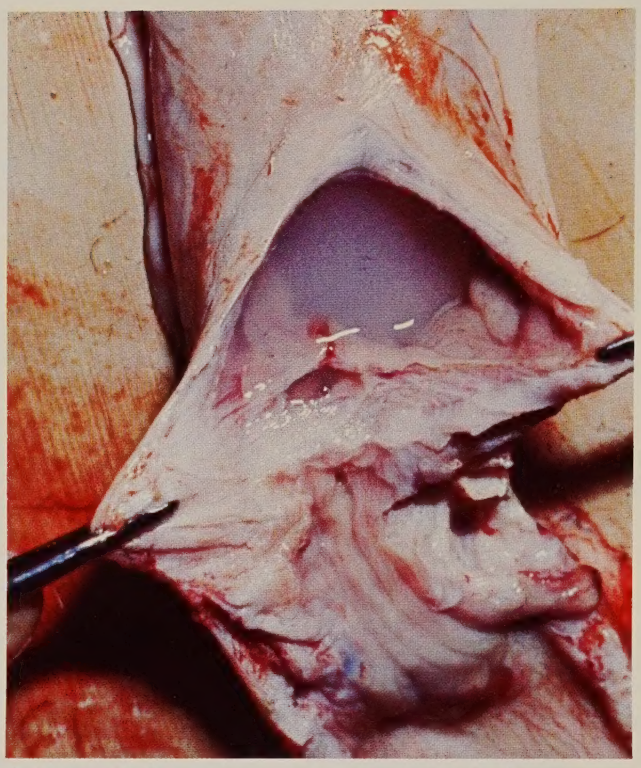
Diagnosis: Clinical disease may be expected during the breeding season; in the Northern Hemisphere this is from February through mid July. Presumptive diagnosis based on finding several mares covered by a particular stallion coming back into premature heat and which appear "dirty" upon vaginal examination. See "Clinical Signs".

In the laboratory, large numbers of polymorphonuclear leukocytes can be found in smears from uterine lumen and in vaginal exudate. Stained with Leishman or rapid trichrome stain, such polymorphs indicate active uterine infection even if bacteriological swabs are negative. Gram negative organisms of various types may be present, as well as the coccobaccillus.

Swabs from mares and the stallion should be streaked on chocolate agar containing 200 to 400 ug/ml of streptomycin plus regular blood agar plates. One plate of each is incubated aerobically and one each anaerobically at 37C for 48 hours. The coccobacillus will grow anaerobically only on the chocolate agar plate; both



3 Congestive inflammation of the cervix (CEM).



4 Accumulation of mucopurulent exudate in the uterus (CEM).

blood agar plates and the aerobic chocolate agar plates should be negative for the coccobacillus colonies. Typical colonies are selected and removed with a fine glass rod for the oxidase test. If oxidase test is positive, a gram stain is performed as well as the catalase tests. If all three are positive a positive laboratory diagnosis is given. If any one of the three is negative, a Cooked Meat Medium is inoculated with the suspect colony and incubated overnight at 37C. From this, new plates are streaked, incubated at 37C both aerobically and anaerobically for another 48 hours and the three tests repeated.

Differential Diagnosis: Two most common vaginal infections in mares causing confusion are those caused by Klebsiella spp. and Proteus spp. Final diagnosis must be made on isolation of the gram-negative coccobacillus.

Collection of Specimens and Laboratory Confirmation: Swabs for culture from mares: All cervical swabs for bacteriologic examination should be inserted in the cervical canal; best taken at estrus. (Cervix may be difficult to penetrate in infected mares). Swab should be guarded to avoid contamination, be rigid enough to penetrate cervical canal. Deposit swab in transport medium. Urethral swab should also be taken. Amies or Stuart's transport media are recommended. Organism can survive for 5 days at 20C in medium, but should be taken to laboratory as rapidly as possible.

Swabs for culture from stallions: Swabs taken from penile sheath, urethral fossa and the urethra. Handle as noted for mares.

Laboratory Culture: follow methods previously given under "Diagnosis."

1-MX-81-6 (280) 7-81

R0000 857261

R0000 857261